PEARL

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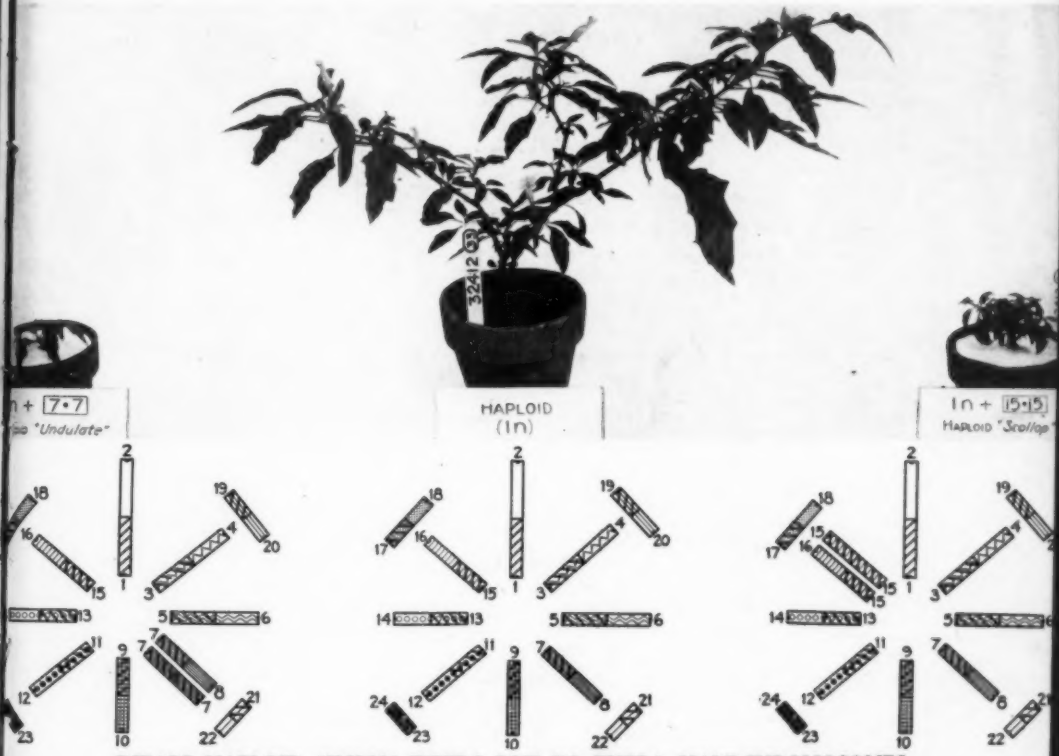

DWARF HAPLOID JIMSON WEEDS DUE TO EXTRA HALF-CHROMOSOMES

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Too Many People—or too Few?

Genetics of the Aquarium Swordtail

Does Appendicitis Run in Families?

"Black-Cutter" Beef Possibly a Genetic Trait

A Pedigree of Ear-Shape Inheritance

"The Blood Group of Diplomacy"

Heterosis in Walnut Hybrids

The Human Aspirin Tablet

VOLUME 28 JUNE, 1937 NUMBER 6

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The

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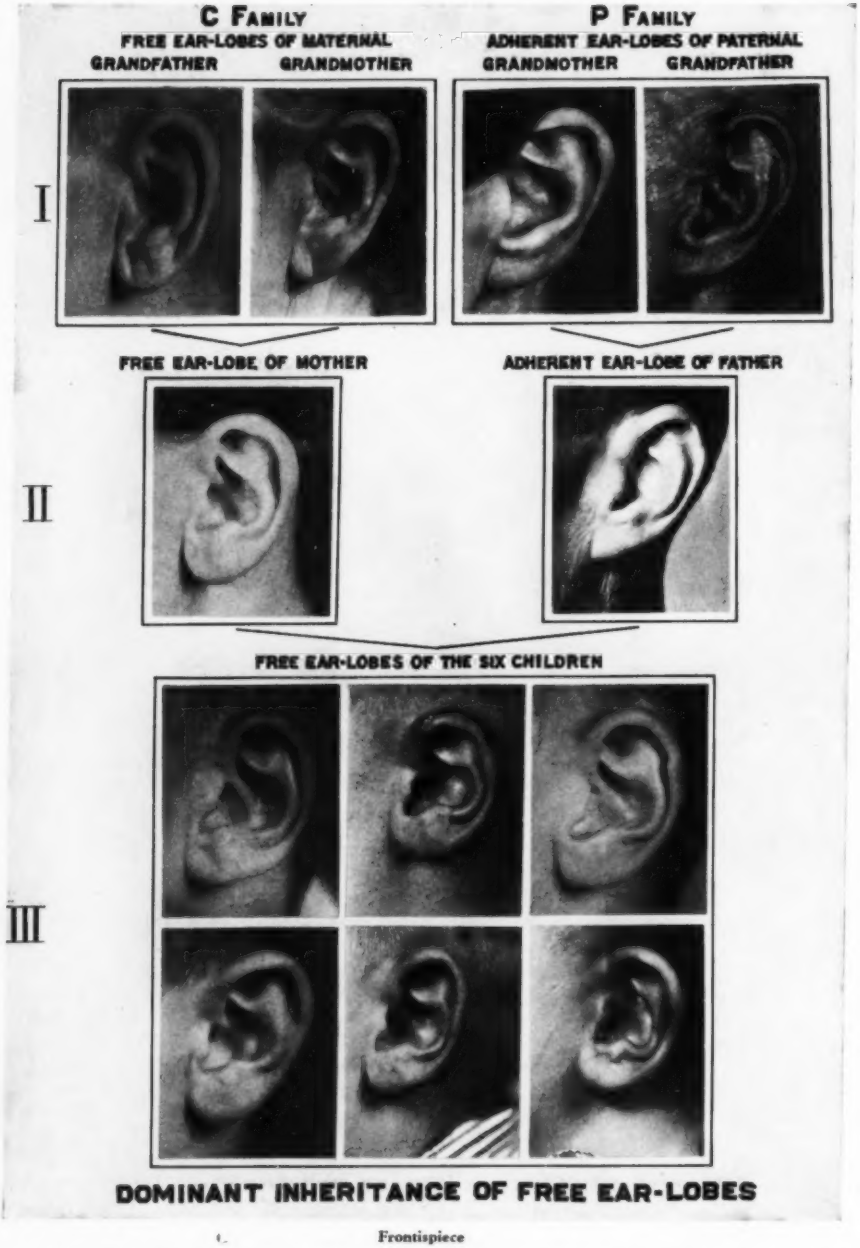


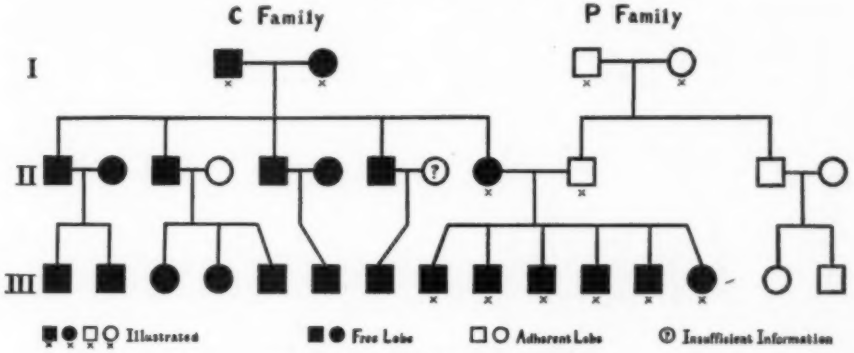
Frontispiece

I, maternal grandparents in the *C* family showing free ear-lobes, and paternal grandparents in the *P* family showing adherent ear-lobes; II, daughter of the *C* grandparents showing free ear-lobes and the son of the *P* grandparents showing the adherent ear-lobes; III, children of the parents (II), all showing the dominant free ear-lobes in the typical Mendelian manner.

EAR LOBE INHERITANCE

An Unusual Three-Generation Photographic Pedigree-Chart

EUGENE F. POWELL and DAVID D. WHITNEY
Department of Zoology, University of Nebraska



INHERITANCE OF EAR-FORM

Figure 1

I, maternal grandparents in the *C* family showing free ear-lobes and paternal grandparents in the *P* family with adherent ear-lobes; II, all children of the *C* family, 4 sons and one daughter, with free ear-lobes; the two sons of the *P* family with adherent ear-lobes. Wives of the sons of the *C* family, two showing free ear-lobes, one with adherent ear-lobes and one doubtful (not observed by authors). Wife of son of the *P* family, not connected with the *C* family, with adherent ear-lobes. III, all of the thirteen grandchildren of the *C* family with free ear-lobes; those of *P* family, with no blood relation to the grandparents of the *C* family, showing adherent ear-lobes.

HILDEN[2] and others have found that free ear-lobes are inherited as dominant characters while Carrière[1] concluded from his studies that they are inherited as recessives. Having studied the inheritance of ear-lobes in three successive generations in the families *C* and *P* and having an exhibit of photographs (Frontispiece) it seems approprite that the striking results should be put on record. This exhibit indicates that free ear-lobes are dominant characters and adherent lobes are recessives.

In the *C* family the maternal grandparents have the free ear-lobes and have transmitted them to their daughter. In the *P* family the paternal grandparents have adherent lobes and have transmitted them to their son. The free ear-lobed daughter married the son having adherent lobes. These parents have six children all of whom have free ear-lobes, thus indicating that free ear-lobes are dominant characters perhaps depending on a single gene.

The mother has four brothers all having free ear-lobes while the father has one brother having adherent lobes. This probably indicates that the maternal grandparents and their daughter are homozygous for free ear-lobes and that the parental grandparents and their son are homozygous for adherent ear-lobes (Figure 1).

The six grandchildren (Frontispiece) comprising the F_1 generation from a homozygous dominant free ear-lobed mother and a father homozygous for recessive adherent ear-lobes show the dominance of free ear-lobes in a typical

mendelian manner. Additional evidence in support of the dominance of the free ear-lobe is obtained from further study of these two families. The character of the ear lobes of all children, their mates, and all grandchildren has been determined, except in one case (indicated in Figure 1) which the authors have not personally observed. The three grandchildren by a son of the *C* family and his wife, who has adherent ear-lobes, show free ear-lobes. This case is similar to that illustrated in the Frontispiece and gives a total of nine grandchildren with free ear-lobes from two similar matings. All thirteen of the grandchildren of the *C* family show the free ear-lobe and provide a striking illustraiton of its dominance. On the other hand, the two grandchildren of the *P* family, whose parents have no blood relationship to the *C* family, show the inheritance of the adherent ear-lobe as a pure recessive.

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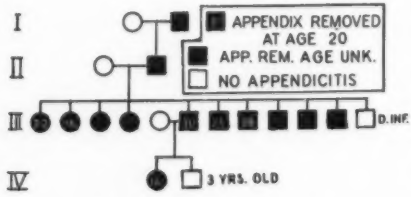
An Appendicitis Pedigree



FOUR GENERATIONS OF APPENDICITIS

Figure 2

IN connection with Dr. Baker's article on the following page, the accompanying fragment of pedigree, showing four generations of appendicitis, has considerable interest. The data on which it is based was submitted by Miss M. L. Nute, of Norwood, Ohio. One of her pupils compiled the pedigree as a result of interest aroused by a "taster test" with P. T. C. paper. In this family the incidence of appendicitis is so high as strongly to suggest some genetic basis. Taken alone a single pedigree would be of very problematical significance and a careful medical check-up would be essential before attempting to arrive at any conclusions. In connection with the two other pedigrees submitted by Dr. Baker it definitely deserves a place in the "record."

It should be borne in mind that it is very unlikely that appendicitis is inherited "as such," as is the case with blond hair, extra fingers, eyes incapable of distinguishing red and green, etc. Genetic differences in resistance to bacterial infection, a peculiar configuration of the appendix, or other factors about which we at present know nothing, probably constitute a predisposition toward the inflammation of this superfluous portion of the human anatomy. The result when expressed in a pedigree chart resembles what we get with the inheritance of a definite morphological or physiological difference, but the chain of events leading up to the "expression" of the "character" has at least one more link of crucial importance—an environmental trigger-stimulus of some kind, more crude no doubt, but fairly comparable to that operating in determining whether a given mouse in a susceptible strain will become a victim of leukemia (see *Jour. Hered.*, April, 1937).—Editor.

A FAMILY PEDIGREE FOR APPENDICITIS

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THE demonstration of a definite hereditary background for functional defects or morbid traits in man is always uncertain since the ordinary methods of experimental genetics are not available and since practically all such defects may also be caused by primarily non-hereditary causes. Therefore every family history that shows a rather definite hereditary aspect is of interest.

Since the beginning of the present century the apparent incidence of appendicitis has greatly increased, due to modern methods of diagnosis, and probably a majority of families have had some experience with this apparently unnecessary organ. However, the family described in this article has such a long-continued clinical history for this defect as to make it seem almost impossible that it could be simply coincidence. All members of this family have been well known to the author for years. Most of them have been cared for by the same physician, whom the author has known for fifteen years and who confirms the statements made herein. The environment of the family has not been such as to make it seem likely that they would have any higher incidence of appendicitis than the population at large.

The "A" Family

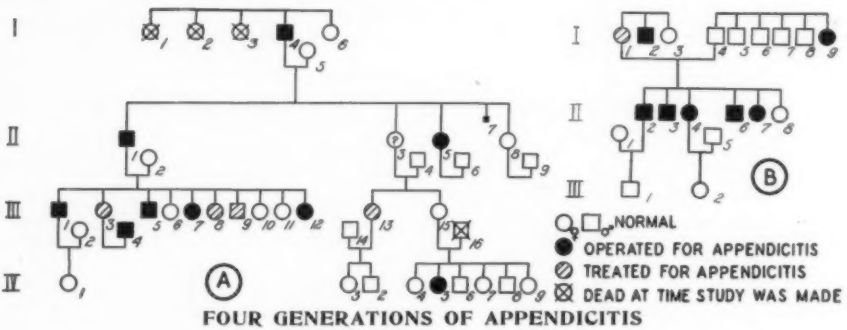
This family has been in the United States for at least seven generations and is a typical mixture of north European stocks. The family name traces to Frankfort, Germany, but very definite and fairly recent English and Irish strains have been introduced. The members of the family have lived either on farms or in small towns of the Middle West for at least five generations and are typical or slightly-above-average middle class citizens of their communities. Most of the members of the last four generations are still living and located in north central Indiana. No members of the family have been so well-to-do as to be able to afford appendicectomy as a fad. The pedigree chart of this family is as shown in Figure 3*A*.

A record of some details concerning each of these cases shows a degree of uniformity as to the nature and onset of the acute condition that would seem to imply some common factor. All except one case have occurred at a relatively young age, and the great majority have become suddenly acute. No attempt has been made to trace the history of the family back of the indicated first generation, as the first member here, I-4, was born in 1859, and appendicitis was not accurately described and diagnosed until after Fitz' medical monograph on the subject in 1886.

The pedigree originates with individual I-4, who was born in 1859 and had his appendix removed in 1933, age 74, following an extremely acute attack and rupture. There is no known previous history of any trouble of this sort. The advanced age at which this case occurred, as contrasted with all the other members of the family, makes it a little less significant than it otherwise might be.

Individual II-1 was born in 1883 and underwent removal of the appendix in 1912, age 29. The case history shows one light attack about a month previous to the acute attack which caused removal.

Individual II-3 is marked as questionable because she is the one member of the family about whom a positive statement cannot be made. She undoubtedly has had a poorer quality of medical care than the other members



FOUR GENERATIONS OF APPENDICITIS

Figure 3

A—Occurrence of appendicitis in the *A* family, of whose members approximately 50 per cent have been operated on or treated for appendicitis. The *B* family is not related but shows an incidence of five cases in a progeny of six, with cases on both sides of the family. Inheritance of appendicitis, as such, is hardly to be reasonably expected. Either a physiological difference in susceptibility to bacterial infection or an anatomical difference in the appendix itself might account for a high incidence of inflammation of this vestigial organ in certain families. These pedigrees suggest that, whatever the causes, a family tendency toward this form of infection does exist.

of the family. Her mother and brother report she had her appendix removed at the same time she had an abdominal operation for ovarian cyst because the doctor "thought" it had been causing her some trouble. She does not know whether this is right or not. The diagnosis is not definite as in the other cases.

Individual II-5 was born in 1889. The ruptured appendix was removed in 1904, age 14, following an extremely acute and almost fatal attack. Unfortunately this branch of the family is now unquestionably without offspring.

Individual III-1, born in 1909, shows a case history of a small amount of chronic appendicitis at the age period from 13 to 15. This then disappeared until the development of a recurrent chronic case in 1934 when the appendix was removed, age 24. This case was not an extremely acute case as were those of the other members of the group, but showed a series of rather uniformly spaced recurring attacks over a period of several weeks preceding removal. As the chart indicates, it is the sibling group of this generation that shows the extremely high incidence of acute appendicitis.

Individual III-3, now 25 years old, has not undergone removal but reports that she suffered more or less constant attacks throughout high school age. Her description is as follows: "I have no way of knowing definitely when my trouble started but I can't remember any trouble before I was in high school when I was about 14 or 15. I was in bed three or four times with acute appendicitis. One attack, much worse than the rest, came the summer before I was 18. I was in bed for a week. Our family physician examined me, found my appendix swelled and told me that it would have to come out sometime. That was my last real attack. I have had a little soreness in that region a few times since." This description, coupled with diagnosis by a reputable physician—who is known to the author and who confirms this statement—makes certain that she suffered from the same condition as did most of the other members of this particular branch of the family but has been simply a little more fortunate in escaping surgical interference. She is now married to III-4, who himself underwent appendicectomy at the age of 21 and whose mother and aunt (mother's sister) both have had removal. Whether there is any really significant

history here the author is, as yet, unable to say. If so, any offspring from this mating should give important data.

Individual III-5, born in 1915, had his appendix removed following a diagnosis of acute appendicitis in 1926, age 10 years. The case history here is the same as that of the majority of the other members of this generation: one very acute attack resulting in removal.

Individual III-6 reports one very light case of abdominal pain at the age of 14. Her parents suspected trouble with the appendix but the trouble passed quickly so was never diagnosed by a doctor. She is now 18 years old and has had no other trouble so is listed as free.

Individual III-7, born in 1919, had her appendix removed in 1933, age 14, following a single very acute attack. The case history is practically identical with that of III-5 and III-12.

Individuals III-8 and III-9 have never undergone surgery but both show histories of very acute cases. III-9, whose present age is 14, had a very severe case when 11 years old. The laboratory report, according to a competent physician, showed a leucocyte count of 22,000 per cu. mm. Removal was avoided by the use of ice packs. In the case of III-8 the attack was less severe, but was definitely acute appendicitis, under the care of a physician. III-8, now 15 years old, was 11 years old at the time. Neither of these individuals has had any further trouble.

III-10 and III-11, at the present time 13 and 11 years old, show a clean history to date.

III-12, born in 1926, had her appendix removed following an acute attack in 1937, at the age of 10 years.

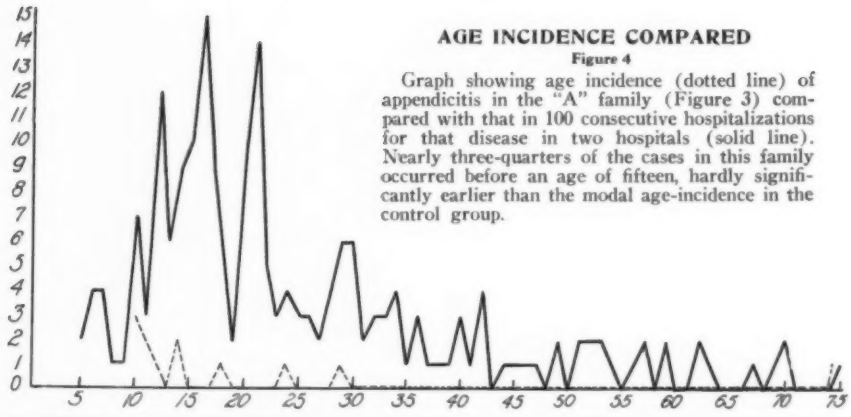
III-13, now 33 years old, had a very decided attack when she was 12. Her mother, II-3, reports that one physician strongly counseled surgery but that the old-time family doctor did not regard it as that desperate and succeeded in carrying her through the attack. She has had no truoble since. III-15 has a clean record in this regard. The author knows nothing of the family record of their respective husbands, III-14, III-16.

All the members of generation IV are still quite young but already one acute case has shown up. IV-1 is less than one year old. IV-2 and IV-3 are 11 and 7 years old respectively. The sibling group IV-4 to IV-9 range in ages from 2 to 14 years. Of this group IV-5 had her appendix removed in 1936. Her age was then 10 years. The case was not so sharply acute as the most of the cases described but had gradually increased in intensity for two weeks preceding the removal.

For the sake of completeness, some mention should be made of individuals I-5 and II-2, since these two as mothers of the two main groups of offspring, could also contribute to any hereditary weakness. I-5 is the sixth of a group of seven—5 females and 2 males. Most of these are now dead and, while the recorded details have not been checked by the author, I-5 herself reports that there never was any trouble with this condition in her family and that in no case could the actual cause of death have been appendicitis. So far as II-2 is concerned, she is one of three siblings, 1 dead and 2 living. Both parents are dead. In all cases the reported cause of death is known and by no possible mistake could it actually have been appendicitis. Also, I am informed by II-2 herself that there has never been any known appendicitis in her entire family. II-2 is the only member of her sib-group having any offspring.

The "B" Family

That this characteristic is hereditary cannot, of course, be stated absolutely, although the pedigree would certainly indicate reason for believing that it is. This case is at least not a totally isolated one. While checking on this one the author has had a similar, though less extensive, pedigree called to his attention. Of a family of six children—II-2, II-3, II-4, II-6, II-7, II-8 in the chart—whose present ages range from 22 to 35 five have had appendicectomies. (See Figure 3*B*). The mother has suffered from more or less chronic appendicitis for years with one or two



AGE INCIDENCE COMPARED

Figure 4

Graph showing age incidence (dotted line) of appendicitis in the "A" family (Figure 3) compared with that in 100 consecutive hospitalizations for that disease in two hospitals (solid line). Nearly three-quarters of the cases in this family occurred before an age of fifteen, hardly significantly earlier than the modal age-incidence in the control group.

moderately acute attacks. The father was killed at the age of 45, but had had no sign of any trouble up until the time of his death. He was the oldest of six children of whom only the youngest has ever had any trouble. I-2, the brother of the mother, underwent appendicectomy at the age of 48. He is the father of 9 children of whom 2 have had appendices removed. The youngest of these nine is now only 12 years old. The age of onset has been somewhat higher in this sib-group than in the one reported on in detail in this paper, having ranged from 19 to 27. This family lived in the same vicinity as the first family until recently; they have scattered in the last five years.

Age Incidence

At the time these facts were first assembled it was thought that the concentration of the ages of onset for the majority of the members of the family might give some added weight to the idea that a hereditary factor was involved. A little investigation, however, shows that this concentration is not any heavier for this family than for the population at large. Through the courtesy of the Culver Hospital of Crawfordsville, Indiana, and the Putnam County Hospital of Greencastle, Indiana, I obtained the age distribution data for the last 100 consecutive cases of appendicitis that have come into each hospital, both of which are typical small city general hospitals of approved rating. Figure 4 shows that data graphically with a similar graph line for this particular family superimposed. There is nothing to indicate that the factors governing age of onset are any different in this particular family than they are generally (see Figure 4).

A little consideration of the percentage of incidence, however, gives added weight to the idea that there is a definite hereditary factor involved. The actual incidence of appendicitis in the general population is a thing on which no accurate statistics seem available. I have made an estimate on the following basis.* The death rate from appendicitis in recent years has ranged from 13.7 to 17.4 per 100,000 population. Most hospitals report a mortality figure of from 4 per cent to 5 per cent of cases treated. This would mean about 300 cases of appendicitis per 100,000 or about 0.3 per cent of the general population annually. If the average length of life is 55 years, this would mean that approximately 16 per cent of the population will undergo hospitalization for appendicitis sometime before they die. This figure makes no allowance for cases that never become acute or for acute cases

*The figures for this estimate were made available through the courtesy of the Literary Research Service of the American College of Surgeons.

that are successfully treated without hospitalization. If we arbitrarily raise the figure to 20% we are making some compensation for this. In contrast with this figure, the four generations of this family contain, at present, 30 people, all hereditarily related, of whom 12 have definitely suffered from appendicitis. This is 40%. If we leave out generation IV, since all the members are less than 13 years old and, therefore, still have a high expectancy of appendicitis, the figures are 11 out of 21 individuals or 52%. On the basis of our general population figure only 4.2 individuals of these 21 should have had appendicitis. For this group S. E. = 1.83 and the actual difference is 6.8 which is 3.8 times the standard error. This gives a probability of less than one chance in seventy-one hundred that the incidence of appendicitis in this family could be simply coincidence.

These results, statistical and observational, can be explained by two different theories. Either a chance factor is responsible and concentration in this family is just coincidence, or some common factor or group of factors—environmental or hereditary—is operating to produce appendicitis in this group. If the factor is chance the family is simply a very rare one; so rare that such an accident should happen less than once in seventy-one hundred families. However, without any especial searching I have been able to find another pedigree for appendicitis, not quite so extended it is true, in the same community. The chances of the same accident occurring twice in the same place and over about the same period of time would seem to be exceedingly small. So the evidence would seem to be against a chance factor. An environmental factor is possible, since most of the cases were in the same family. However, not all the members were raised at the same time, and there does not appear to be any great or specific environmental difference from other families in the vicinity that are known to have little or no appendicitis. The idea that there is a definite hereditary factor or factors operating would seem to be the most plausible.

Whether this hereditary cause is a unit factor or some multiple factor arrangement would be impossible to determine on the basis of the meager data available. With the observations that in three generations slightly over half of the individuals have shown the defect, that in the one sib-group 70% have shown the defect, and that in a second case a sib-group has developed the defect in 83% of its members a unit factor dominant hypothesis would be plausible. However, the number of cases is still much too small to make a positive statement as to the type of heredity. The exact nature of the heredity and the question of whether it is widespread in the population must await the collection of more data. Blacker[2] and Bauer, Fischer and Lenz[1] both cite pedigrees showing apparent dominant transmission of gastro-intestinal troubles, both of a structural and a functional nature. Since the immediate cause of appendicitis is usually bacterial infection, it is impossible to say exactly what weakness may have favored the onset of such a condition. Some peculiarity of structure or location, unusually poor blood supply, a very poor connection between the appendix and the rest of the digestive tract all suggest themselves as factors which might enable the bacteria, usually present in all the lower digestive tract, to be so troublesome in the vermiform appendix in the individuals belonging to the family here described.

The record of this family has been filed with the Eugenics Record Office and will be followed up.

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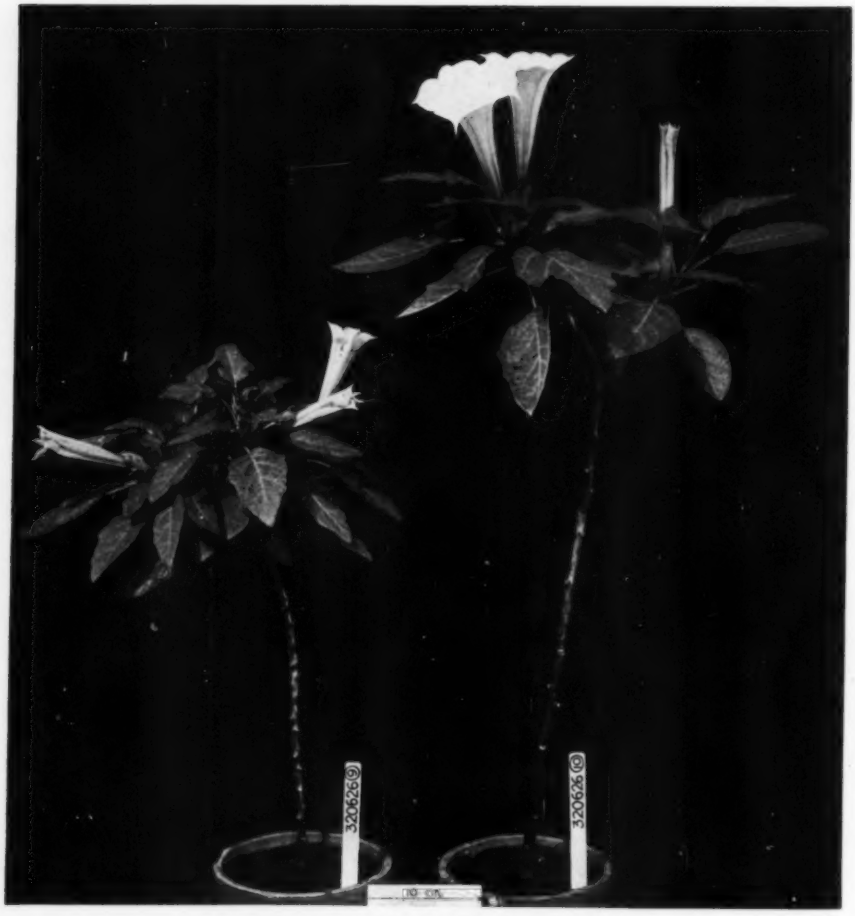

HAPLOID AND DIPLOID DATURAS

Figure 5

Potted plants of *Datura metel*. On the left a haploid ($1n$); on the right a diploid ($2n$) from the same race. Note the smaller flowers and narrower leaves of the haploid. Haploids have only a single set of chromosomes, instead of the paired chromosomes usually found in the tissues of higher plants and animals, and are consistently less vigorous than diploids. The smaller flowers, leaves, and stature of this haploid are typical of these forms.

BALANCED AND UNBALANCED HAPLOIDS IN DATURA

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SINCE the first haploid in Datura was reported in 1922[8] the behavior of its chromosomes has been described in two publications.[1,2] Photographs of pot-grown plants and of plants grown in the garden have already been presented in other publications.[7,5] The internal anatomy in comparison with that of the other balanced types and of types with single extra chromosomes has been considered elsewhere.[16] The occurrence of mutations from our first haploid was discussed in an article in this JOURNAL in 1927.[9]

It is the purpose of the present paper to summarize the occurrences and breeding records of haploids in Datura since our last report ten years ago and to discuss a number of unbalanced haploids which have occurred in our cultures.

Since 1921 we have to date recorded 211 haploids. In the last six years (1930-1936) we have grown approximately 410,000 plants to a stage at which haploids could be determined. Among these we have listed 73 haploids, which is 0.018 per cent. It might be said that the haploids are so distinct that they can usually be readily distinguished by inspection. It has been usual, however, to confirm the determination by an examination of the pollen. Haploids show between 80 and 90 per cent aborted pollen grains and those that are good are all of the same size and like those of normal plants. In rare cases when the haploidy involved a race or a species different from our standard, the chromosomes have been counted in pollen mother cells.

According to some investigators, haploids are likely to occur as a result of a species cross. This does not seem to be the case in Datura. Among 2,707 F_1 plants from species crosses, we have had only two haploids (0.074 per cent). These two occurred in 1923 in two crosses with *D. stramonium* as the female parent pollinated with *D. ferox* pollen. The two haploids were pure *D. stramonium*. One of the *stramonium* parents was a $2n + 11 \cdot 12$ primary type and the other was a $2n + 7 \cdot 7$ secondary type. In both cases apparently a $1n$ egg developed parthenogenetically.

Of the 211 haploids which we have identified, 109 have occurred among the offspring of crosses of various kinds, including both interspecific hybrids and *inter se* crosses, and 102 from selfs and open pollinated capsules. These figures do not tell directly whether haploids are more frequent from selfs or from crosses without a tabulation of the number of offspring from selfs and from crosses during the period in which haploids have been listed. Such a tabulation has not been made for the complete period. In two years, however, — the winters of 1934-35 and 1935-36, and the summers of 1935 and '36,—we had 76,080 plants recordable from selfs, among which were 13 haploids, and 77,356 plants recordable from crosses, among which were 14 haploids. These records give a mutation rate of 0.017 per cent haploids from selfs and 0.018 per cent haploids from crosses. There is no evidence from these figures that haploids are any more likely to come from crosses than from selfs.

Of the 109 haploids from crosses, 47 could have come from the female parent only, as was determined by the genetic constitution of the two parents. Regarding the remaining 62 haploids from crosses, it could not be determined positively that the haploid came from the

PRUINOSA HAPLOIDS APPROACH DIPLOID IN VIGOR

Figure 6

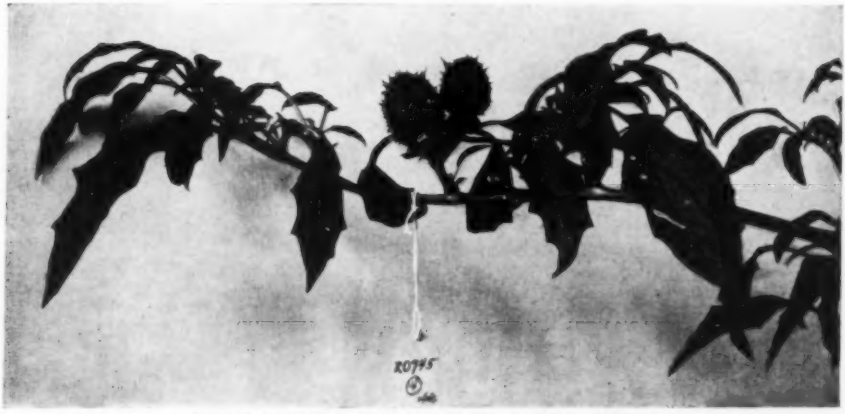
Field-grown plants of *Datura pruinosa*. On the right a haploid and on the left a diploid. The finer foliage of the haploid is evident, but in this species the haploid apparently is not so distinct a type as in *D. stramonium*.

female parent, either because both parents carried the same genes or because one or both were heterozygous.

Of the haploids which we have recorded, 209 came from *D. stramonium*, one from *D. metel* (Figure 5) and one from *D. pruinosa* (Figure 6). The reason that *D. stramonium* has such a relatively large number is due primarily to the fact that we are growing many more plants of this species than of other species. Furthermore, we are more familiar with the appearance of haploids in *D. stramonium*. As a matter of fact, the haploid in *D. pruinosa* was not recognized as such at first but was later recorded as being an off-type and probably a haploid from the very high proportion of aborted grains observed in its pollen. A more careful examination of the plant showed that it had the characteristics of a haploid,—narrow leaves and sterile flowers,—and a count of its chromosomes showed it to be in fact a haploid plant. In this species apparently the appearance of haploids is not so striking as in *D. stramonium* and in this and in other species we may have missed certain instances of haploids in our cultures.

Our first haploid was identified among plants in an experiment in which the flowers of the parents had been subjected to cold treatment. Among 6,612 such plants there were 4 haploids, which is a percentage of 0.06. In view of the number of haploids which have been obtained without the cold treatment it is not believed that there is any connection between the treatment in these early experiments and the occurrence of haploids.

The first haploid, which was number 20745(4), was kept growing for six or seven years by grafting it onto normal diploid stock. Several times during this period normal $2n$ branches arose on the haploid. One such case is illustrated in Figure 7 in which two relatively large capsules are shown on a short side branch on a part of the plant that has obviously haploid leaves. The capsules of haploids usually have no seeds at all, though occasionally they may have one, two, three, or, rarely, more. The reverting branch shown in Figure 7 arose in 1926 when the haploid plant was five years old. This branch was determined to have 12 pairs of chromosomes by study of its pollen mother cells. A haploid in 1927, 27956(11), also had a branch which was determined to be $2n$ by the small amount of pollen abortion and on which was produced a normal $2n$ capsule. A plant in the summer of 1933, 320330(41), was on one side haploid and on the other side diploid. The line of demarcation between haploid and diploid tissue went through the middle of one leaf and also through one of the



FERTILE DIPLOID BRANCH ON HAPLOID

Figure 7

Branch from the first known haploid among flowering plants. The short branch with two large capsules is $2n$ while the earlier growth of the plant was all $1n$. Doubling of chromosomes to form sectors of $2n$ tissue has occurred in other haploids of *Datura* and in other species.

flowers. Although this plant was approximately half $1n$ and half $2n$, it is believed it started as a haploid and reverted to a diploid on one side at an early stage in its development. Since the reversion to diploid recorded for the first haploid Datura plant,[4] sectorial reversions of a similar kind have been found in an unbalanced haploid type to be discussed later and also have been reported by other investigators[12, 15] for haploids in other species.

Harland working with cotton[11] found that of twin seeds within the same seed coats one was almost always a haploid and the other a diploid. Kappert working with flax[13] also found a number of cases in which a twin pair consisted of a haploid and a diploid. In Datura of five twin embryos, both of which were grown to a recordable size, we have found only one case in which one of the twins was a haploid and the other a diploid. The determination in this single case was checked cytologically.

In our last paper on haploids, published in 1927,[9] we reported twelve $2n + 1$ types (3.05 per cent) from 393 F_1's from our first haploid, 20745(4). Since that time we have grown nearly twice as many F_1's from the same haploid and are able to add eleven more $2n + 1$ types to the records. Our total records now stand as follows: 1,137 F_1 plants of which 23, or 2.02 per cent, were $2n + 1$ types; two were tetraploids and 14 were indeterminable. Our records regarding the number of $2n + 1$ types in F_2's from haploids and in later generations are as follows: total plants recordable—33,928, of which 96 (0.28 per cent) were $2n + 1$ types; two were haploids; 25 were tetraploids; and 25 were indeterminable. It would seem, therefore, so far as the records go, that $2n + 1$ types are more likely to occur in the F_1's from haploids than in the offspring of diploids.

Mutations

In our last paper on haploids[9] we reported four recessive gene types which appeared in the F_2 generation from our first haploid. These were *curled* (*c*), *tricarpel* (*tc*), *early* (*e*), and *short stamens* (*ss*). They appeared in the offspring of 173 F_1 individuals and give a mutation rate of 2.31 per cent. Since this first report we have tested 417 more F_1 plants from the same haploid and have obtained four new mutations—namely, *albino-2*, *pale-3*, *glaucous-4*, and

NORMAL HAPLOID COMPARED WITH EXTRA CHROMOSOME FORMS

Figure 8

Two 1*n* + 1 types, with unmodified haploid (1*n*) in center. Below each plant is shown a model of its chromosomes. The two "secondaries" each have a different "double-half" chromosome added to the haploid complement of twelve chromosomes. When it is considered that the primary haploid (1*n*) is a relatively weak plant, the unbalancing effect of two extra chromosome halves added to the set of twelve is obviously very great.

Male Sterile-1. This gives a mutation rate for the second group of 0.96 per cent. All told, we have tested 590 F_1 individuals and found eight of them were heterozygous for new genes. This total gives a mutation rate of 1.35 per cent. It was earlier mentioned that $2n$ branches were produced on our haploid. Of the offspring of one of these $2n$ branches, 72 plants were tested by selfing. One of them was found to be heterozygous for a new gene—*albino-3*. The number of spontaneous mutations in Datura is relatively rare. In our last article we said that the offspring of 362 plants in the F_2 or later generations from the haploid had been tested and found not to contain new genes. Since this time in F_2 and later generations from the first haploid we have found only three plants that were heterozygous for new visible characters — namely, *pale-18*, *sickly-1*, and *broadleaves-1*. It is difficult to figure out the mutation rate that this number of gene mutants would represent since not all the plants were grown to a stage at which certain types of gene mutants would be noticed. It is safe to say, however, that they represent at the very least 1,000 individuals adequately tested by selfing, and the number may run up as high as 5,000 or 6,000. The very highest mutation rate that could be assigned to these mutants from diploid plants is 0.30 per cent and the actual mutation rate is probably considerably lower. It appears to be the case, therefore, that our first haploid had a higher mutation rate than diploids, although this was less marked in the later part of its life. What the cause may be for this evident increased mutation rate we have not been able to discover. It is not connected with aging of the seeds, which is known to be a cause of increased mutation rate. One of the parents heterozygous for a mutated gene (*c*) came from seeds only three weeks old. Five others came from seeds six months or less old, one came from seeds seven months old, and only one came from seeds as old as eight and a half months. We do not suggest that the increased mutation rate in the haploid investigated is due either to the condition within the plant associated with its being a haploid or to some unknown environmental condition associated with keeping it alive for several years, most of which time it was kept going as a grafted individual. We are leaving the records as we have found them: they indicate a considerable increase in both chromosomal and gene mutations which presumably took place within the haploid plant.

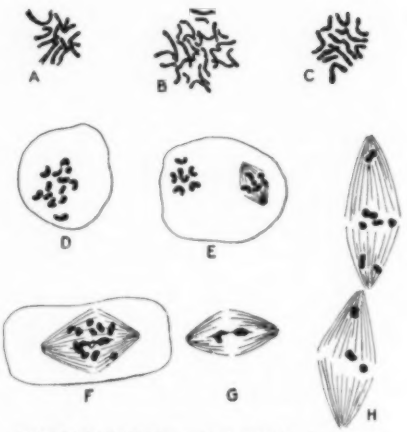
Unbalanced Haploids

We have so far discussed the occurrence and breeding behavior of balanced haploids. Among the 211 plants which we have listed as haploids in our cultures, four were apparently unbalanced haploids since they are recorded as probably $1n + 1$ types. One of these, 320246 (49), came from a compensating type[6] with the formula $2n - 1 \cdot 2 + \cdot\ 1 + 2 \cdot 9$ which had been crossed to a diploid male. Our records do not suggest which chromosome was extra. Another probably unbalanced type, 33116(4), came from a cross between the compensating type $2n - 5 \cdot 6 + 5 \cdot 5 + 6 \cdot 19$ and a diploid male. It was recorded as probably $1n + 5 \cdot 5$. We were unable to confirm cytologically our tentative determination of these two plants as $1n + 1$ types before the plants died. In two other cases it was possible to study the chromosomes and to be sure that they both were in fact unbalanced haploids. A brief report of these two types has already been made[10]. The two types in question are represented in Figure 8 in 4-inch pots along side of the haploid of comparable age in a 5-inch pot. Beneath each of these three haploids is given a model which represents our interpretation of its chromosomal constitution. The size of the modified haploids indicates their extreme weakness of growth when it is borne in mind that the unmodified haploid in the center is a relatively weak-growing plant in comparison with diploids.

A preliminary determination of the chromosome number in the first plant was made from root tips and in the second plant from buds. All the subsequent work was done on grafted material except in the case of the root tips, which

were taken from the original plants. On account of the difficulty in finding pollen mother cells in division and the need of depending in part on divisions in the somatic tissue of the buds, the usual acetocarmine method was not used. Instead, material was fixed in Lewitsky's or modified Carnoy's solutions sectioned, and stained in iron hematoxylin.

The potted plant 32602(3) shown on the left in Figure 8 appeared spontaneously among offspring of a secondary $2n+7\cdot7$ individual which had been crossed to prime type No. 50.[3] It was recorded by appearance as probably $1n + 7 \cdot 7$ and was extremely weak. Despite attempts to keep it alive by grafting, it died a few months after the photograph was taken. The buds which were formed dropped before the proper stage of development had been reached and thus neither pollen grains nor dividing pollen mother cells could be obtained from them. Cytological examination was limited to somatic tissue in the buds and in the root tips. The number of chromosomes in the somatic tissue was 13. Figure 9*A* shows a cell from a root tip at metaphase with 13 chromosomes. The number of dividing cells in the sections was small, probably due to the slow growth of the plant, but in every case the cells both of the central cylinder and of the cortex yielded counts of 13 chromosomes. That the plant was a $1n + 1$ type was established but, since unfortunately no pollen mother cells nor megaspore mother cells were observed, its classification from appearance as a $1n + 7 \cdot 7$ could not be confirmed cytologically. Its female parent was the secondary $2n + 7 \cdot 7$ which would be expected to have $1n$, $1n + 7 \cdot 7$, and $1n + 7 \cdot 8$ female gametes. These by parthenogenesis might give rise to haploid offspring. Gametes with extra chromosomes other than $7 \cdot 7$ or $7 \cdot 8$ would be extremely rare from a parent of this constitution. Of the two chromosomes most likely to be an extra in a female gamete with the given parentage, the $7 \cdot 8$ chromosome can be eliminated from consideration as the extra in our modified haploid. When present as an extra in $2n + 1$ types it causes distinctive peculiarities in the appearance



CHROMOSOMES OF TWO $1n + 1$ PLANTS

Figure 9

A—Chromosomes from root tip of $1n + 7\cdot7$ plant; metaphase plate showing 13 chromosomes. *B*—Root tip: metaphase plate showing 26 chromosomes. This is a diploid cell arising from a doubled $(1n + 1)$ cell. *C*—Nucellar tissue: metaphase plate showing 13 chromosomes. *D*—Pollen mother cell at first metaphase: 11 free plus 2 attached univalent chromosomes. *E*—Pollen mother cell at second metaphase: one group contains 6, the other 7 chromosomes. Such pollen mother cells, not having a complete set of chromosomes are not functional. *F-G*—Megaspore mother cell at first metaphase: (*F*) 11 free plus 2 attached univalents. The "doughnut" shaped secondary chromosome (15 • 15) is the right hand member of the pair drawn alone in *G*. *H*—Megaspore mother cell at second metaphase: 4 chromosomes on the lower spindle, 9 chromosomes on the upper spindle. Some chromosomes lag at the equatorial plate. (*A-B*, 1220×; *C-H*, 1000×.)

of the plant which were not apparent in our $1n + 1$ type. Furthermore, the unbalance due to this chromosome is so great that $2n + 7 \cdot 8$ types are weak plants with extremely poor viability. It is doubtful if a plant could survive the increased unbalance which would result from adding the $7 \cdot 8$ chromosome to a haploid. All the evidence available, therefore, favors considering our 13 chromosome plant to be a secondary $1n + 7 \cdot 7$ type.

The plant 32412(74) shown on the right in Figure 8 was one of two haploids which appeared among the offspring of

a secondary $2n + 15 \cdot 15$ heterozygous for prime type No. 20 which had been backcrossed to our standard Line 1. It was considerably more vigorous than the other $1n + 1$ type and by grafting was kept alive for over a year. Its flowers had either no pollen grains at all or had about 85 per cent aborted grains. Many attempts to self the plant failed even when pollinations were made by hand. Furthermore, no seeds could be obtained in crosses with diploids when the haploid was used either as a male or as a female.

As material for cytological studies, root tips and buds of various sizes were used. Chromosome counts were made in somatic tissue and at the first and second metaphase in the megaspore mother cell and pollen mother cell. Special attention was given to the behavior of the 13th chromosome in meiosis. The development of the embryo sac and of the tetrads and microspores was also followed.

A study of microsporogenesis and of male gametogenesis in haploid Daturas was made by Belling.[2] A comparison of the observations on our $1n + 1$ plant with those made by him show that these processes do not differ essentially. Since this same is true of the megasporogenesis it is not necessary to describe them in detail here.

Counts made in pollen mother cells and in megaspore mother cells invariably showed the presence of 13 chromosomes. At the first metaphase the 13 univalents lie scattered on the spindle (Figure 9*F*). In a few cases two chromosomes were connected by ends forming a chain of two univalents (Figure 9*D*, *F*). It is undoubtedly the presence of the 13th chromosome which causes this connection. Its behavior will be discussed later. The univalents move at random to the poles and after a short interphase form two nuclei. After the second division is completed, four nuclei of variable size are formed, two being usually smaller, two larger. Their size depends on the previous distribution of chromosomes which varied considerably. Figure 9*E* shows the distribution of $6 + 7$ and Figure 9*H* shows the distribution of $9 + 4$.

A detachment of chromosomes at first anaphase is not rare. It leads to a formation of microcytes. Cases of non-reduction were also observed; they probably account for the small amount of good pollen in anthers and of normal-appearing embryo sacs in ovules. One peculiarity observed in ovules might be mentioned here. It is the large number of twin embryos within single ovules which were found in some young buds in early stages of megasporogenesis. Twinned embryos have also been found in $2n$ and $3n$ plants, but much less frequently than in this haploid.

Let us see what evidence is available regarding the chromosomal constitution of this $1n + 1$ type. It was the offspring of a $2n + 15 \cdot 15$ plant which was heterozygous for prime type No. 20. This prime type has the modified chromosomes $11 \cdot 15$ and $12 \cdot 16$ instead of the Line 1 chromosomes $11 \cdot 12$ and $15 \cdot 16$. The parent plant therefore had, in addition to ten bivalents, the five chromosomes $11 \cdot 12$, $11 \cdot 15$, $12 \cdot 16$, $15 \cdot 16$, and $15 \cdot 15$. The $1n + 1$ offspring must have gotten three of these. Viable gametes might have contained the following groupings in which the $15 \cdot 15$ chromosome was included:

1)	2)	3)
$11 \cdot 12$	$11 \cdot 15$	$11 \cdot 12$
$15 \cdot 16$	$12 \cdot 16$	$12 \cdot 16$
$15 \cdot 15$	$15 \cdot 15$	$15 \cdot 15$

The following combinations which do not contain the $15 \cdot 15$ chromosome were also possible:

4)	5)	6)
$11 \cdot 12$	$11 \cdot 12$	$11 \cdot 15$
$15 \cdot 16$	$15 \cdot 16$	$12 \cdot 16$
$12 \cdot 16$	$11 \cdot 15$	$15 \cdot 16$

7)
$11 \cdot 15$
$12 \cdot 16$
$11 \cdot 12$

The appearance of the plant ruled out combinations (4) and (7). An attempt was made to determine cytologically which of the remaining five combinations were in the gamete which produced the $1n + 1$ plant. Among bivalents a secondary chromosome is recognizable because of its tendency to bend back on itself to form a "doughnut." This method of identification, however, is not easy among univalent chromosomes, which themselves are short and more or less bent. In Figure 9*G*, one

TETRASOMIC PLANTS SHOW EFFECTS OF CHROMOSOME UNBALANCE

Figure 10

The effect of adding an increasing unbalance of the *21 • 22* chromosome is shown at *A* and *B*, which are respectively a potted plant of a simple trisomic ($2n + 21 \cdot 22$) and of a tetrasomic [$2n + (21 \cdot 22)_2$]. The tetrasomic has a chromosomal unbalance of *2* extra chromosomes against *24*, the number in a normal diploid. The simple trisomic has numerically half as great unbalance (1 vs. 24). *C*—Field-grown tetrasomic $2n + (15 \cdot 16)_2$; and *D*, a simple trisomic $2n + 15 \cdot 16$.

of the two attached chromosomes looks like a "doughnut," strongly suggesting that the 15 • 15 chromosome was present. And because the doughnut was connected with another univalent, combinations (1) and (2) are the most likely ones. In addition to the 15 • 15 chromosome, combination (1) contains the chromosomes of Line 1 and combination (2) contains the chromosomes of prime type No. 20. Our conclusions are based on the assumption that like ends of chromosomes are attached. The 15 • 15 chromosome could be attached to either the 15 • 16 or the 11 • 15 chromosome. The exact condition could be determined by an examination of the chromosome arrangement in the offspring. Unfortunately, however, the plant was sterile and no offspring could be obtained.

As was mentioned in the preliminary report[10], the cells of the somatic tissue

contain 13 chromosomes. Numerous counts were made later with additional material which showed that this number was constant in the somatic cells of the buds (Figure 9*C*). But all counts made on all the root tips examined (a total of 7) showed 26 chromosomes (Figure 9*B*). The cells in the cortex and in the central cylinder of the roots were $2n + 2$. This plant, in other words, was a chromosomal chimera; it had haploid buds, both in somatic and sporogenous tissue (29 buds examined), but had diploid root tips. It is not known in what tissue this doubling occurred. Since the plant was very small and looked like a modified haploid rather than a modified diploid, it is probable that the plant started as a haploid and portions of it became diploid later. A more extensive investigation of root tips of this plant might have disclosed some that were haploid or some that were partly diploid and partly haploid, forming thus a sectorial or periclinal chimera. Doubling of chromosomes in roots of haploids is probably not uncommon. Of three unmodified Datura haploids which were studied cytologically, one was found to have $2n$ roots (4 root tips examined). Similar cases have been reported by Ruttle[15], Hollingshead[12], and others, and sectorial reversions of haploids to diploids in branches of Datura have been mentioned earlier in the present paper.

The number of new cases of haploidy in plants is increasing rapidly. So far haploids have been represented in more than a dozen different genera. The presence of extra chromosomal material in haploids, however, must be rare. Lesley and Frost[14] in 1928 found a Matthiola plant which had an extra fragment in addition to the 7 univalents—the haploid number of chromosomes. It is not unlikely that in the offspring of $2n + 1$ Daturas other unbalanced haploids have appeared, but have been unable to survive due to the extreme weakness brought about by the extra chromosomes. It should be remembered that the amount of unbalance exercised by a single extra chromosome is dependent upon the chromosomal constitution to which the extra chromosome is added. Photographs of capsules of diploids and tetraploids modified by the presence of a single extra $21 \cdot 22$ chromosome have been published in this JOURNAL[6] and elsewhere and it has been pointed out that the unbalancing effect of the extra chromosome in a $4n + 1$ type is relatively slight in comparison with the unbalancing effect of the same chromosome in a $2n + 1$ type. In the former case the unbalance, if expressed numerically, is 1 against 48; in the latter case, it is 1 against 24. The present paper gives an example of a new unbalance brought about by a single extra chromosome—namely, 1 against 12.

In the diploid series we have two tetrasomic $2n + 2$ types in which the same chromosome is represented twice as extras. These are $2n + (21 \cdot 22)_2$ and $2n + (15 \cdot 16)_2$. Photographs of the capsules of the former tetrasomic have been published in the article cited[6]. A potted plant is shown in Figure 10*B* alongside of a simple trisomic $2n + 21 \cdot 22$. Figure 10*C* shows a field grown plant of the tetrasomic $2n + (15 \cdot 16)_2$ beside a simple trisomic $2n + 15 \cdot 16$. In both of these photographs the relative weakness of the tetrasomic in comparison with the simple trisomic is evident. The tetrasomics have the same numerical unbalance — namely, 2 against 24 — that is found in a $1n + 1$ type which is 1 against 12. It should be remembered, however, that the haploid, to which the extra chromosome was added in the plants described in the present paper, is already much weaker in growth than a diploid. Hence, the unbalanced $1n + 1$ haploids may be expected to be much weaker than the corresponding tetrasomic diploids. The extreme unbalance which we have just discussed in our two tetrasomics is due to the addition of unmodified primary chromosomes. It happens that the two cases of $1n + 1$ types so far determined were probably due to the addition of a double-half, secondary chromosome. In these modified haploids, therefore, we had a half chromosome represented three times, — two times as extra material. The nearest comparable unbalance in a diploid due to secondary chromosomes was brought about by crossing a $2n + 2 \cdot 2$ female plant with prime type No. 5 in which the

11 • 12 chromosome has been replaced by a 2 • 11 • 12 chromosome. Offspring were obtained which were $2n + 2 \cdot 2$ heterozygous for prime type No. 5 and hence carrying an additional • 2 half. Such a plant, therefore, had an unbalance of three extra • 2 halves and was proportionately modified in appearance and weakened in growth.

Summary

From the spontaneous occurrence of over 200 haploids in Datura it is shown that they are no more likely to occur as the result of a wide cross than as the result of selfing and that their occurrence is not stimulated by cold treatment. Several of the haploids have borne branches in which the chromosome number was doubled. About 2 per cent of the F_1's from our first haploid were $2n + 1$ types, which is a chromosomal mutation rate of nearly ten times that from comparable $2n$ parents. Nearly 600 F_1 individuals from this same haploid were tested by selfing and 8, or 1.4 per cent, were found to be heterozygous for new genes. This gene mutation rate from the haploid is 5 to 30 times as great as in comparable diploids.

Four plants have been recorded as $1n + 1$ types, of which two have had the chromosomes counted. One was probably $1n + 7 \cdot 7$. The other was probably $1n + 15 \cdot 15$ since it came from a $2n + 15 \cdot 15$ female parent and the 15 • 15 chromosome could be identified in pollen mother cells of the + 1 haploid. It apparently began as a $1n + 15 \cdot 15$ seedling but its roots changed to $2n + (15 \cdot 15)_2$ by chromosome doubling. The extreme unbalance in these modified haploids (1 vs. 12) is numerically comparable to the unbalance (2 vs. 24) in the tetrasomics $2n + (15 \cdot 16)_2$ and $2n + (21 \cdot 22)_2$. The $1n + 1$ forms have a lessened vigor due to the initial weakness of the haploid to which the extra chromosome is added.

AUTHORS' POSTSCRIPT — Since correcting proof, a new $1n + 1$ plant has appeared in offspring of a $2n + 13 \cdot 13$ parent. Root tips and somatic cells of buds showed 13 chromosomes. In pollen mother cells were 11 univalents plus 1 bivalent. Latter consisted of two primary 13 • 14 chromosomes, not of 13 • 14 plus 13 • 13. Cytological findings as well as appearance of plant indicate that it is a primary $1n + 13 \cdot 14$ type. Our records therefore now include both primary and secondary $1n + 1$ types.

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HEADED FOR THE LAST CENSUS?

I. Overpopulation or Underpopulation?—A Review of Conflicting Opinions

GUY IRVING BURCH

"RACES and nations, like individuals, are born, grow old and die."

When Dr. Corrado Gini, director of the Statistcal Institute of Rome, presented before the guests of the Harris Foundation the view epitomized in the above[1] his learned audience was inclined to smile. But as things have since shaped up in the "family of nations" and in our own backyard, many scientists have begun to wonder if there wasn't some truth in what the eminent Italian said. Yet hope springs eternal even in the breasts of statisticians.

It has long since ceased to be "news" when some writer compares life to a stage on which we human beings are merely puppets. But who besides Dr. Gini has thought of comparing whole races and nations to skyrockets and lesser fireworks, which flicker or flare high for a moment in the history of our race, but always fall to the cold earth and to oblivion? He says:

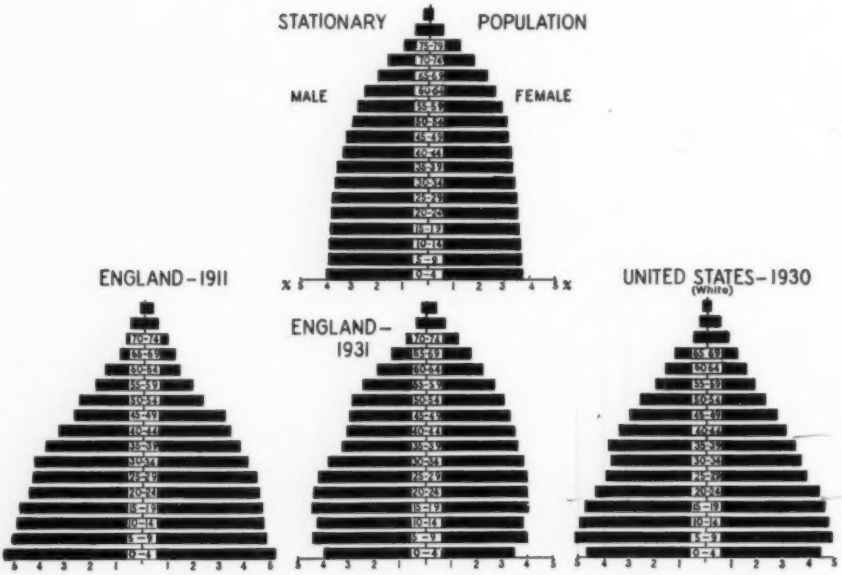
> Thus the world may be compared to an immense field of fireworks; each rocket representing a (national or racial) stock which rises until it touches a different height, it shines brilliantly and sometimes explodes to illumine with its light a large part of the sky, then falls and extinguishes itself to leave room for other rockets which are rising from below, they, too, to sparkle in their turn with a more or less diffused but always a fleeting light.

In this grand display of fireworks, we Americans might wonder in which direction our own "star-spangled" rocket is pointed. Very likely Dr. Gini would say that we are about at our height. On this question, however, Dr. Robert R. Kuczynski of the London School of Economics might give us some information. What he says in his many books on this general subject is far from encouraging and he is very fond of the phrase "bound to die out." He lumps us together with central and northwestern Europe, as members of that growing group of nations in which "the population no longer reproduces itself." This is the equivalent in his estimation of saying that we will soon be on our way down-and-out.[2]

At this point we may strenuously protest. We may point out that our birth rate is still some five points higher than our death rate and that our inhabitants have increased about 20,000,000 during the last sixteen years. Furthermore, a glance at our athletic records, our declining illiteracy and death rates, and our increasing average length of life, should be enough to convince almost any "reasonable person" that the American "race" is on the "up and up." If further evidence of our national strength is needed our great wealth of natural resources and our unparalleled progress in industry, science and invention, should suffice.

The "True" Birthrate

To all this the scientific Sherlock Holmes might reply: "*Superficial,* my dear Watson. Your population increase is now an *illusion.* Your record low death rate is *fictitious.* Your increasing literacy among the stupid has put a premium on low grade literature and your newspapers and magazines now are published for ten-year-old minds. Athletic records are made by a highly trained minority and do not truly represent the vast majority of the population which is mostly a stoop-shouldered, flat-chested, city-dwelling lot whose increasing urban life is not only ruining the nation's health but upsetting its economic and social conditions as well. You are becoming slaves of your industrial machines, rather than their masters. And as for your increasing average length of life brought about by medical science helping the weaklings through their dis-



AGE DISTRIBUTION—"CHRISTMAS TREES"

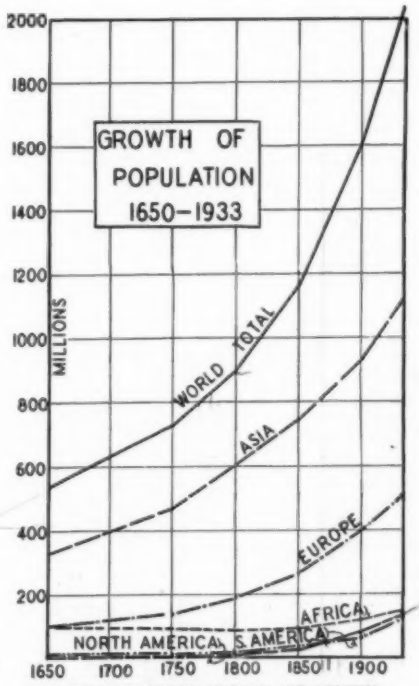
Figure 11

The percentage distribution of the various age groupings (0-4 years, 5-9 years, etc.) gives a "Christmas tree" of varying outline depending on the birth rates and the death rates. In the stationary population each group is larger than the group below it and smaller than the group above. Where the population is not reproducing fast enough to furnish the necessary replacement, the lower age groups contain fewer individuals than those above. Only in recent years have there been more children aged 5-9 in the United States than there are children aged 0-4 years of age. Modified from Lorimer and Osborn, *Dynamics of Population* (stationary population and U. S. 1930); and from data in Carr-Saunders, *World Population*, Oxford, 1936 (England 1911 and 1931).

eases, that is producing an older and weaker race. Finally, do you know that those least fit for either natural conditions or complex civilization are rapidly out-breeding the fittest?"

"Incredible," we may say. But no, we are told that it is really elemental knowledge to those who make it their business to understand the census records. Then we may ask what is the "true" situation as regards, for example, our population growth and our record low death rate? The fact that the present birth rate (17 per thousand) exceeds the death rate (11 per thousand) seems to prove that we can't be anything but a growing population. "So what," we next ask, "is all this statistical blue funk which gives the experts the jitters?"

The reasoning of the experts is rather simple, and quite disconcerting. Because of our much higher birth rate of the past and the recent arrival of large numbers of young immigrants, Dublin and Lotka[3] point out, we now have an unusually large proportion of our population between the ages of 20 and 45. Now as deaths tend to be few and blessed events tend to be many among persons between the ages of 20 and 45 years, the country "has" a lower death rate and a higher birth rate than we would expect with a "normal age distribution" of population (that is, the distribution be expected in a population with stable age groupings with no immigration). For that reason these statisticians insist that under such conditions our "record" low death rate would be above 16 per thousand population instead of 11, or a "true" death rate higher than our "true" birth rate. Since our present surplus of young adults



WORLD POPULATION GROWTH

Figure 12

From 1650 to 1933 the population of the world increased from slightly over half a billion to over two billion. This growth curve, closely approximating a geometrical progression, is the basis for the claim of the "overpopulationists" that the world is too crowded now and in serious danger of becoming more so unless means can be found to check the present *increase* of about 27 million a year.

cannot be maintained by present birth rates, this is what is meant by saying that our population growth is an illusion and our record low death rate fictitious.

Now, when the large proportion of our people now in the "favorable" ages moves along to the older ages in which deaths are many and births are few, we may expect to find our death rate rapidly increasing and our birth rate rapidly declining. Consequently, we shall ere long seen to be hastening toward the last census,—or at least toward a census in which many fewer noses will be counted.

Were it merely a matter of fewer people, that might easily be arranged for, or even welcomed with open arms, but the problem is not so simple. Unfortunately, a decline of numbers brought about by a continuously declining birth rate, especially when medical science is increasing our average length of life results in an aging and an "enfeebled race." As a matter of fact, old age is already coming rapidly upon the American "race." To illustrate, in 1850, at about the beginning of the gold rush to California, only one American in ten was over the age of fifty years; today one out of every five is beyond that age, and Dublin estimates[4] that by 1980, forty-three years hence, as much as one-third of our population may be past the half century mark. In the meantime, the broad-headed Slav and slant-eyed Mongolian races give little evidence of lowering their exceptionally high birth rates which keep their populations young. Furthermore, recent studies[5] show that among those races the fittest are still breeding rapidly and there is little medical science to preserve the lives of those lacking the inherent vigor to survive. These conditions are quite a contrast to those in *enlightened* Western Civilization as we shall presently see.

From all this we may gather the impression that our civilization is like a man skating on thin ice which has already begun to crack. The only escape, thinks Kuczynski,[6] is to pivot as quickly as possible and to head back in the direction of the high birth rates of the past. But when this "way out" is suggested, there are any number of scientific authorities in this country ready to cry: "No! No! It is the high natural increase rates of the past which have made us over-heavy with population that in reality is causing the ice to crack." If this be true, we find ourselves on the very sharp horns of a troublesome dilemma.

World Overpopulation

If Gini and Kuczynski have painted a dark picture of our future because of the declining birth rate, Dr. Raymond Pearl of Johns Hopkins University,[7] and a number of other American scientists,[8] have produced just as gloomy a

masterpiece of statistics which go to show that the high natural increase rates of the past are the *cause* of our present perilous condition. Pearl's argument runs as follows: For every single square mile on the land area of the earth's surface there are now living more than forty human beings. When we realize that this includes mountain tops, deserts, tropical jungles, unhealthful and undependable climates, and vast expanses of land periodically visited by great natural disasters such as earthquakes, volcanic eruptions, floods and increasing erosion, it is a sobering thought. This means that upwards of 2,074,000,000 of human beings are right now struggling to get a living on our inexpansible planet. To make the situation worse, about half of this great number of people are living on about one twentieth of the land area of the earth. This also suggests that the other 95 per cent of land is either not of much value for sustaining human life or that it is unavailable because of immigration restrictions. Dr. Pearl puts the finishing touches on his picture by saying that each year the world, already sorely burdened by troubles, many of which are due to population pressure, takes on a net load of about 27,000,000 *new* human beings, while the chemical elements that sustain life are being exhausted at a rate of billions of tons annually.

"Mass-suicide" is the measure that man usually takes when increasing numbers press too desperately, Dr. Pearl warns,[9], for he has discovered that this tendency to mass-suicide is a principle in animal life which extends from microbes to men. Like the Norwegian lemming, many species, including man, first increase slowly, then rapidly, and finally become so agitated because of density of numbers that they "explode." In the case of the lemming it starts running and travels on pell-mell until it plunges headlong into the sea to a watery grave. Man considers this action of the lemming the "limit of dumbness," says Dr. Pearl. But, he says, man does much the same thing and for much the same reason when he marches into war.

Perhaps Dr. Pearl has never studied stock market psychology—the kind they have in Wall Street—to gather data for his theory of mass-suicide. But in man it would appear that the same psychology that recurrently forces customers to "buy at the top" and "sell at the bottom" in the stock market is much akin to the psychology that leads nations to destroy themselves. It is that irresistable faith which seems to force men to believe that what *is* always *will be*. In the early 1920's the stock market started up, and with a few relatively minor setbacks it kept right on marching, until by 1928 and most of 1929, it just "couldn't go down." About 1630 the population of Western Civilization started to increase. It increased slowly at first, then faster and faster, until at the beginning of the twentieth century it just "couldn't stop increasing." Through these centuries we have acquired a "booster" psychology which now seems to be a part of our very natures. The repeated warnings that populations were reaching the "exploding stage" were cried down, much the same as were the repeated warnings about the inflated stock market in 1928 and 1929. The result is that we have now reached a stage of population inflation where we are afraid if countries of Western Civilization increase further they will "explode" and we are afraid that if they lower their birth rates the race will become old and tottering.

Optimum Population

We spend much time amusing or exciting ourselves about *quantity* of population, but as Professor A. B. Wolfe of Ohio State University, reminds us[10] *quality* of inhabitants is of equal importance, and that the one cannot be successfully separated from the other in the consideration of this vital question of what constitutes an optimum population. Whatever views we hold regarding this important matter, we run into theories or dogmas which now seem to be a vital part of man's nature. One of these deep-rooted "feelings" is that the inhabitants of a country should continue to keep right on increasing until that country contains all the people it "can hold" or "can support." Judging by this test of

the number of people a nation should have, no country on earth is "overpopulated." This is a risky principle to accept as a guide, Dr. Wolfe believes, holding further that it is safer and more reasonable to consider that a country is overpopulated *when it contains more people than is conducive to the best interests of the inhabitants of that country.* Judged by this test, there are very few countries on earth that are not overpopulated at the present time.

If the countries of Europe contained the number of people which was conducive to the best interests of the inhabitants, why have Europeans flocked to this country in such large numbers during the nineteenth and early part of the twentieth centuries, or why has history been filled with accounts of one people pushing into the territory held by others? Professor Maurice R. Davie of Yale University reminds us that the relationship between population and land tends to be lower in the immigrant-receiving countries than in the emmigrant-exporting countries. As a consequence of this relationship this country, which for a century has been the great immigrant-receiver, has been "more democratic, its people enjoy greater social and political equality, there is more individual freedom of conduct, traditional and legal restraints are fewer, military burdens are lighter, and there is a greater latitude for religious belief and practice." [11]

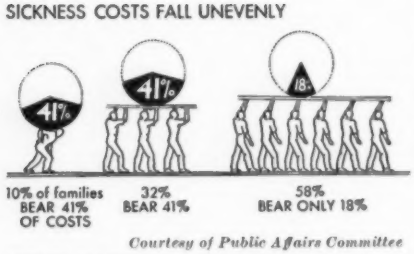
It is well known that this country is the *richest* country in the world and politicans like to refer to it as containing the "highest standard of living the world has ever seen." Professor Vladimir Woytinsky in a report just published by the International Labor Office at Geneva[12] tells us that at the beginning of the depression, the national income of the United States made it "richer than all the European countries combined" and "the sum of the income of the people of the United States constitutes approximately half of the world's income." This statement is probably near the truth,* but even our general standard of living is not a great deal to brag about if President Roosevelt is correct when he says he sees "one-third of a nation ill-housed, ill-clad, ill-nourished." [13] If this is the situation in a country where a democratic government "guarantees" equa rights under the law, and equality of economic opportunity is the ideal, what must the situation be in the rest of the world which contains sixteen times as many people but only about the same total wealth and income as is enjoyed by this country?

"Go *East* Young Man"

If it can be shown that even the United States is beginning to contain more people than is conducive to the best interests of its inhabitants, there should be little argument about other and more densely inhabited countries with far lower standards of living. For decades the advice has been "Go West, young man, go West," and for three centuries population has moved toward the Pacific Coast in a continuous stream. Up until 1930 the inhabitants of California increased in a record-breaking fashion. But between 1930 and 1935, the Census figures indicate that the population of California decreased, suggesting that economic opportunity there was diminishing. For the first time in the history of the United States, border patrols have been established by several of the western states which allow only those with adequate resources to act on Horace Greeley's well worn dictum. No doubt, this situation is temporary but it indicates which way the wind is blowing, and that a great change is taking place in migratory trends and in economic opportunity in the United States.

After a three year study[14] of interstate migration and economic opportunity in this country the experts of the *Study on Population Redistribution* led by Dr. Carter Goodrich of Columbia University, tell us that the year 1932 "may be taken by future economic his-

*A single American corporation paid more in Federal and State taxes than the entire tax revenue of several of the smaller European countries (Am. Rept. of Std. Oil Co. of Indiana, 1936).—Ed.



Courtesy of Public Affairs Committee

WHO PAYS FOR MEDICAL CARE?

Figure 13

How much do medical costs *lower* the birthrate of the two-fifths of the population which pays four-fifths of the total doctor's bill? How much does nearly free medical care *raise* the birthrate of the three-fifths who pay only one-fifth of the collective doctors' bill? The exact answers to these questions are unknown at present, but that such differentials have no effect on birthrates is almost inconceivable.

torians as the end of the era of pioneering abundance and the beginning of a time when resource depletion was to affect materially the national life." These scientists and many others insist that our timber, mineral, and other natural resources are being exploited at a dangerous rate, and that erosion by wind and water has begun to destroy millions of acres in this country. They estimate that there may be as many as 6,000,000 people too many in the Southern States for their best interests, and also that the cut over regions of the Great Lake States and the prairie Great Plains States are now overpopulated according to the "best interest" test. Where are these supposedly surplus people to go? No great economic opportunity is to be found for them in agriculture, while in industry we are suffering from what is thought by many people to be a chronic state of unemployment. This may be drawing the picture a bit too dark, but to say that the United States is underpopulated, as many Europeans are wont to do (perhaps as propaganda for migration) demands a rigorous definition of the term "underpopulated."

Broadly and humanely speaking, the "optimum point" would seem to be the number of people in a country that is conducive to their best interests. "The conclusion is inescapable . . ." says Dr. John D. Black of Harvard University, and formerly chief economist of the Federal Farm Board, "that at least some sections of the United States are well past the optimum point, and perhaps all of them are past it, in the present stage of the arts." [15]

It has often been said that our chief problem is one of distribution. This statement is correct as an ideal. But better distribution requires an intelligent and high quality population, and is there any evidence which indicates that the quality of our inhabitants is improving? First let us examine the physical and then the mental health trends.

We have mentioned that our health has been labeled artificial. This view has been given wide currency by no less a medical authority than Dr. Alexis Carrel, eminent surgeon and Nobel Prize laureate.[16] In reply to President Roosevelt's recent inquiry as to how engineering schools might help solve the nation's problems, a Georgia Tech faculty committee headed by Professor Montgomery Knight[17] asserts that "in saving the lives of the weak and physically unfit, we are in a real sense, undermining the physical well-being of future generations." This committee report goes on to say that "fully recognized by authorities in biology and medicine is the decadence of our national health through the advance in medical science." This observation tallies well with Carrel's views, and represents the considered opinion of a rather imposing group of competent authorities.

During the World War those who had charge of our national defense were considerably surprised to find that nearly half of the drafted men (a group in the prime of life) were found to have one or more notable physical defects. Since the war our physical health does not seem to have made much progress as judged by the number of patients in general hospitals. In 1935 there were three persons admitted to general hospitals to every two that were treated in such hospitals in 1922, according to figures from the Federal Census and the American Medical Association.[18] Furthermore, the

days of medical treatment in such hospitals have nearly doubled, increasing in round numbers from 53 million to 95 million. Incidentally, even in 1922, long before the New Deal for the "forgotten man," less than half the days of treatment were paid for by the patients and about one-third of them were presented to the patients absolutely free.

The rapid increase of patients in hospitals for mental disease is perhaps an even more serious problem. Where there was one patient in institutions for mental disease in 1880, there are now (1934) about ten, according to Census records. The population of these institutions has increased nearly four times as fast as the population of the country. This rather alarming increase in the number of patients in such hospitals, according to Pollock and Malzberg,[19] of the New York Department of Mental Hygiene, may be explained in part, *but only in part*, by the more general use of hospitals in recent years. It is estimated by reliable scientific authorities that even yet, fewer than one-tenth of the mentally defective are housed in state institutions.

Before considering further the trend in the mental growth of the American people let us briefly inquire into a possible cause of the increase of mental defectives and the so-called "unfit." For some time past it has been the policy of the most civilized peoples to have mercy on the unfit that nature would have destroyed with little ceremony, and that under cruder and harsher conditions would have been eliminated in human society. This has resulted in saving the lives of many of the weak, the dull, and the mentally defective who formerly would not have lived to reproduce their kind. Certainly this is a humane and laudable policy, but when our benevolent society permits, and even indirectly encourages these unfortunates to increase their kind with compound interest, the situation is fraught with peril. "Philanthropy which has esablished hospitals and institutions to prolong the lives of the unfit may be at times of doubtful value. . . . The result has been a protected race, and some day the protection may find itself inadequate and the race will be deciminated if not doomed," [20] says Dr. G. B. Cutten, President of Colgate University.

"Is it not therefore apparent, in the light of the evidence here presented," says Dr. Earnest A. Hooton,[21] president of the American Association of Physical Anthropologists, "that modern man is selling his biological birthright for a mess of morons; that the voice may be the voice of democracy, but the hands are the hands of apes?"

To remedy this situation Dr. Hooton[22] recommends a biological purge: "we must either do some biological housecleaning or delude ourselves with the futile hope that a government of the unfit, for the unfit and by the unfit will not perish from the earth."

Dr. Franz Boas[23] of Columbia University, the eminent German-American anthropologist, takes issue with Dr. Hooton on this subject. Dr. Boas does not question the "fact that the number of subnormals admitted to institutions is increasing," that "an increasing number of individuals are found to be unequal to the strenuous tempo of city life," and that "in some cases hereditary unfitness has been established and for these sterilization is justifiable." But, he says, "it is quite impossible to hazard even a guess as to whether the intelligence of a *whole* population is increasing or decreasing" (Italics ours).

"Though the *average* intelligence may remain the same," says Dr. Boas, "the range from the lowest to the highest in the scale has been expanded." It would seem difficult to prove this, but if it is true, it would appear to create serious problems for a democracy.

"In a less highly concentrated environment," he says, "many of the same individuals might lead quite satisfactory lives * * * since social and economic environment are important factors in determining the behavior of an individual." But what is causing this "highly concentrated environment?" This brings us back to the unfortunate affects of population and economic pressure upon the quality and behavior of people.

The humane interference with nature's process of evolution is thought by many

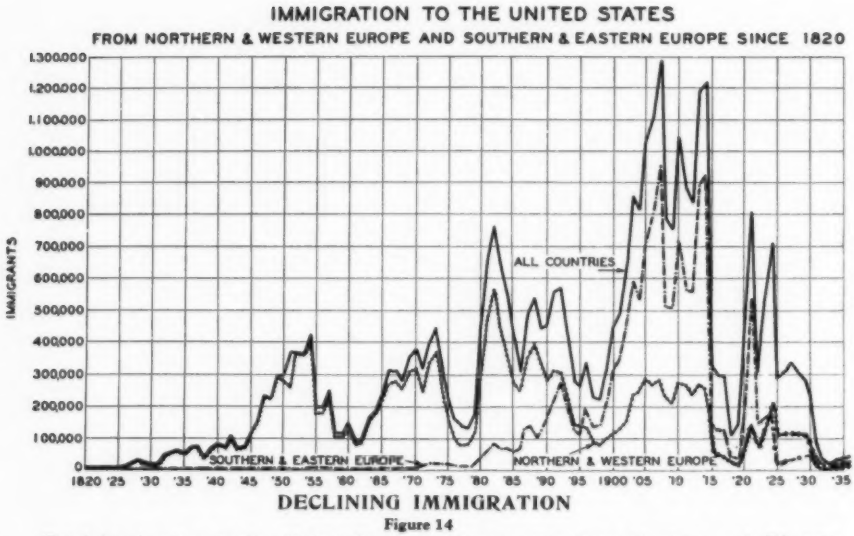


Figure 14

Total immigration to the United States and immigration from Northern and Western Europe and from Southern and Eastern Europe, from 1820 to 1936. Economic and other considerations have produced a profound change in the last quarter of a century. In 1907 an all-time "high" of nearly one and a third million immigrants entered the United States; in 1936, only 36,329 were admitted. These figures do not consider the departures from the United States, which during the last two or three years exceeded immigration for the first time.

students of the problem not only to have greatly increased the unfit, but also to have hindered the increase of the fit. Dean Inge, of St. Paul's, London, points out that "Every unwanted baby, kept alive by humanitarian interference, drives another baby out of the world or prevents him from coming into it." [24] Carrel[25] holds that "the abnormal prevent the development of the normal." According to the Malthusian principle of population if fewer people with bad heredity were born, people with good heredity would automatically respond by an increased birth rate. This is essentially Gresham's *law of economics* that "bad money drives out good money" applied to population growth. Is there any reason to hope that this same principle may prove any less disastrously effective in debasing our human coinage?

While this humanitarian policy has been in full swing for generations among our native population, the problem of mass immigration has further complicated the situation. At various periods in American history it has been heatedly contended that immigration was causing a deterioration of our citizenry. Practically all of the founders of the nation opposed mass immigration,[26] such as this country experienced between 1840 and 1924, and even Thomas Jefferson predicted the most disastrous results if the nation permitted it.

During the early settlement of this country interference with nature as regards the survival of the weak and dull was near a minimum. When the savage and the wilderness faced the newcomer, and before the steamship made trans-Atlantic travel easy even to the invalid and the imbecile, it was hard sledding for even the fittest physically and mentally to survive. Out of whole shiploads of people who had the courage to leave Europe for America, sometimes relatively few survived to rear any children in this country. Half of the passengers in the *Mayflower* died within a year of the time she left England. Nine-tenths of the settlers of Jamestown died

or went home during the first two years.[27] It was not until about the middle of the nineteenth century — more than 200 years after the sailing of the *Mayflower* — that life became fairly safe and easy throughout the vast expanse of this continent, and by that time the steamship had made the Atlantic passage relatively comfortable.

In the 1840's the first big wave of immigration struck these shores. It was caused not by any great suffering from "underpopulation" in this country, sparse as the inhabitants were, but by poverty-stricken people fleeing starvation and famine in lands abroad. According to the Irish Census of 1851, "The great mass of emigrants from Ireland is composed of the poorer classes who, being withdrawn from the population, must diminish the proportions of the illiterate, of persons occupying fourth class house accommodations, and of those dependent on their own manual labor for support."[28] Writing on Irish emigration in the authoritative work *International Migrations*,[29] D. A. E. Harkness, of Queen's University, Belfast, says that the emigration resulted in "a reduction of 356,000 or nearly three-fourths in the number of single-room mud cabins, the dwelling houses of all three superior classes increased in number."

It has been contended on Darwinian principles that the descendents of the colonists and early settlers would be much more highly select physically and mentally than later arrivals who did not have to go through rigorous selection even to reach America. Thus, Dr. Alexis Carrel, himself a newcomer from France, has this to say:

> The descendents of the founders of American civilization may still possess the ancestral qualities. . . . The women belonging to the oldest stock, whose children would, in all probability, be of good quality, and who are in a position to bring them up intelligently, are almost sterile. It is the newcomer, peasants and proletarians from primative European countries, who beget large families. But their offspring are far from having the value of those who came from the first settlers of North America.[30]

It is of more or less academic interest to speculate at this late date on what kind of people there would have been in this country had there been no immigration after the United States was established as a nation. In their recent book[31] on the Huntington family in America, Ellsworth Huntington and Martha Ragsdale say that "the facts set forth in this book suggest that in many ways life would be more satisfactory . . . if the United States had received no immigration after the colonial period." These writers conclude that this country now would have a population of substantially the same size and of a higher quality. They suggest that the average buying power and standard of living might be one-fourth higher, that the percentage of home-owners might be increased by half, that criminality and social dependency (which costs this country about $20,000,000,000 annually) might be reduced by half, that the population might contain a far higher percentage of people able to complete a college education, and that the power of the demagogue and the political boss might be greatly diminished. Even though the conclusions of these authors were to be discounted by a half or more, it seems clear that the colonial population missed a great eugenic opportunity when they failed to build a tight immigration wall around what was to be the United States.

It should be remembered, however, that it is hazardous to apply general conclusions to individuals, as the brilliant careers of such newcomers as Steinmetz, Baekeland, and Carrel amply illustrate. Some of the finest and most capable people in this country are new arrivals and have proved their ability in a short time. Also it is incorrect to imagine that all who came to this country before the steamship were physical and mental giants. Some of them were of low quality, if we may judge by the criticism of their contemporaries and from the "progeny test" of their descendents in this country today.

Whatever may have been the effects of immigration upon the American "race" since the coming of the steamship, American scientists and laymen alike are now generally agreed on a

policy of immigration restriction and selection. Investigations of population problems in this country such as those made by the highly qualified experts of the *Study on Population Redistribution* and the *National Resources Committee* should have placed this question beyond reasonable doubt. But whether our ancestors came to this country in a sailing vessel or in a steamship, we are all in the same boat now. Nevertheless, the problem remains, of the kind of people we will be even so soon as a generation hence, and even farther in the future. This question is a very live one because, while we are all "in the boat," we are a pretty diverse lot, and we reproduce at very diverse rates. A baby is just as much an immigrant biologically as a new arrival from across the seas. What do the statisticians tell us about the kind of babies we are welcoming to our shores, who are destined to be the men and women a generation hence?

(To be Concluded)

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WHAT CAUSES "BLACK CUTTER" BEEF?

A Historical Basis for the Hypothesis That the Condition Known as "Black Cutter" in Beef is Caused by Hereditary Factors

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IN the beef trade a condition is more or less frequently encountered in which the cut surface of the meat does not brighten normally. Such carcasses are commonly designated as "dark" or "black cutters." Evidence has been presented that dark beef from young, well-finished cattle is after roasting, as attractive, tender, and palatable as beef that was bright colored before cooking. Because, however, of its dark appearance it is merchandised at materially lower prices than normal beef and consequently the cause of the condition is of considerable interest in the packing industry.

The freshly cut surface of normal beef is dark, similar to venous blood, due to muscle hemoglobin. Upon exposure to the air the cut surface brightens rapidly as the hemoglobin changes to oxyhemoglobin. According to Mackintosh and Hall,[7] this proceeds rapidly for 30 minuates, but continues for a period of three hours at a decreased rate. These authors for convenience divide dark cutters into two classes although there is no definite demarkation between them: (a) dark cutters or those in which the color fails to brighten materially or more slowly than normal, and, (b) black cutters or those in which the color does not brighten at all.

The investigations of Mackintosh and Hall[7] further show that there is a positive correlation between total hemoglobin content of the tissues and the brilliance and chroma values of the meat, but that the total hemoglobin content of black cutting beef was within the normal range. They found that black cutting beef, which even when ground failed to brighten perceptibly, changed to bright red when placed in an atmosphere of oxygen under pressure. Since hemoglobin in the absence of oxygen is dark purple and oxyhemoglobin is bright red they conclude that some condition exists in the tissues of black cutters which renders them impervious to the oxygen of the air.

Alleged Causes

In the meat trade, age, exercise, excitement, feed, delayed bleeding, peculiar pigmentation, and heredity are factors commonly mentioned as contributing to or causing dark beef.

It is generally recognized that there is a progressive darkening in the color of beef through the range from veal to old, mature animals caused by an increase in muscle hemoglobin. The degree of finish or fatness has also been shown to affect the brightness of the meat.[1,6,7] These factors, however, cannot account for the appearance of black cutters in high quality, well-finished yearling and two-year-old cattle.

Exercise throughout the fattening period and violent exercise and excitement before slaughtering has not resulted in dark cutting beef under controlled experimental conditions.[3,4,7] In experiments at the Illinois Experimental Station,[5] there was some indication that bleeding, delayed up to 10 minutes after stunning of the animals, caused darker colored beef. The results from year to year were, however, inconsistent and no effect of delayed bleeding for a similar period has been found by Hinman[3] and by Mackintosh and Hall.[7] Since delayed bleeding may result in less complete bleeding, the muscle hemoglobin might be augmented by blood hemoglobin and thus affect color to some degree. This

would not be expected to change the permeability of the tissue to oxygen, however, as Mackintosh and Hall have shown in the case of dark and black cutting beef. Thus far all attempts to influence color by kind of feed used during the fattening period have given negative results.

Since it has not been possible to produce dark or black cutting beef under experimental conditions by any of the means discussed, a hereditary basis would appear the next logical field of inquiry. Representatives of the packing industry, with whom the author has discussed the subject, have encountered these carcasses from time to time for as long as they can remember. The condition apparently appears sporadically without any definite relation to age, feed, origin or handling of the cattle. These men state that the dark color may appear in some muscles of the carcass and not in others. This was the case in a "dark cutter" recently observed by the author. The eye of beef (longissimus dorsi) was nearly black while the fillet (psoas major) had a normal, bright red color. Similar variations in the muscles of the round have also been described.

Early Observations

Although hereditary factors have been suggested as the cause of dark and black cutting beef, insofar as the writer is aware, no evidence has been presented. A historical basis for a genetic explanation of this anomaly was presented in a book by George Culley.[2] This book was published in 1807, but the footnotes indicate that the observations recorded go back at least to 1774.

With reference to the distribution of Longhorn cattle in Lancashire, Leicestershire, Warwickshire, Gloucestershire, Cheshire, etc., Culley states (page 57), "The kind of cattle most esteemed before Mr. Bakewell's day, were the large, long-bodied, big-boned, coarse, flat-sided kind and often lyery or black-fleshed." No mention of lyery or black flesh is made regarding the Galloways or Kyloes of Scotland but it is stated that none of the Alderneys were in the least subject to black flesh.

In his discussion of the "Shorthorn or Dutch Kind" and the probability of their Continental origin, Culley states: "I remember a gentleman of the county of Durham (Mr. Michael Dobinson), who went in the early part of his life into Holland in order to buy bulls; those he brought over were of much service in improving the breed; and this Mr. Dobinson and neighbors even in my day were noted for having the best breed of short-horned cattle, and sold their bulls and heifers for very great prices. But afterwards, some other persons of less knowledge going over, brought home some bulls, that in all probability introduced along that coast the disagreeable kind of cattle, well known to the breeders adjoining the river Tees, by the appellation of lyery, or double-lyered; that is, black-fleshed; for, notwithstanding one of these creatures will feed to a vast weight and though fed ever so long, yet will not have one pound of fat about it, neither within or without, and the flesh (for it does not deserve to be called beef) is as black and coarse-grained as horseflesh."

With further reference to Shorthorns he mentions: "This breed, like most others, is better and worse in different districts; not so much, I apprehend, from the good or bad quality of the land, as from a want of attention in the breeders. In Lincolnshire (which is the farthest South that we meet with any number of this kind of cattle) they are, in general, more subject to lyer, or black flesh, than those bred farther north; and in that rich part of Yorkshire called Holderness, they are much the same as those south of the Humber, of which we have been speaking. It is probable that they had either stuck more to the lyery black-beefed kind, than their more northern neighbors, at that unfortunate period when they were imported from the Continent, or that the latter had seen their error sooner. But, from whatever cause this happened, it is a fact that as soon as we cross the Yorkshire Wolds northward, we find this breed alter for the better; they become finer in the bone, in the carcass, and, in a great measure, free from that disagreeable lyery sort

which has brought such an odium upon this (perhaps) *most valuable breed.*"

No reference is made to black beef in the brief discussion of the cattle of Herefordshire. Culley says, however, that the Shorthorns and the Longhorns were in possession of the best and greater part of the Island and that they had been variously intermixed in different parts of Great Britain. This statement indicates the possibility of rather wide distribution of stock that may have carried the blood of the "disagreeable, black-fleshed kind."

These definite historical references to black flesh make it desirable to take advantage of any opportunities to determine more definitely the incidence of black cutters in commercial cattle and to ascertain the relative prevalence of the anomaly in breeds and strains of purebred cattle, with the ultimate aim that the hereditary hypothesis may be subjected to critical experimentation.

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INHERITANCE OF OKRA-LEAF AND ROUND-LEAF IN UPLAND COTTON

A Note on Brown's and Cotton's Data

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In a recent issue of the Journal of Heredity, Brown and Cotton[1] reported a mutant in Upland cotton (*Gossypium hirsutum*) which they designated as "round-leaf." First generation hybrids between this mutant type and a normal broadlobed type "showed none of the specific round-leaf characters," and the F_2 generation segregated in the proportion of 31 normal plants to 15 round-leaf plants. In addition, one plant which "appeared to be somewhat intermediate" was observed but no explanation was attempted. Neglecting this one plant, the proportion of the normal to round-leaf plants does not differ significantly from a simple 3:1 ratio since the probability (according to Warwick's[7] tables) of a fortuitous deviation as great or greater than the observed is .15. The first generation hybrid of the cross round-leaf by okra-leaf also showed no round-leaf characters. Six different classes were recognized in the F_2 generation of this cross, one of them the normal broad-leaf form which they state was "not expected."

Numerous workers, including Carver[2], Harland[3], McLendon[4], Peebles and Kearney[5], and Ware[6] have reported an intermediate condition in crosses between okra-leaf and broad-leaf and three distinct classes in the F_2 generation in the monohybrid ratio of 1 okra-leaf : 2 intermediate:1 broad-leaf. This indicates a case of monohybrid character with incomplete dominance. The data of Brown and Cotton seem to indicate simply an interaction of two genes (or factors). Using Harland's[3] designation O_o for okra-leaf and O_n for normal, and assigning the symbol *rl* to round-leaf and

Rl to its normal allelomorph, the genotype of the okra-leaf parent is written: O_oO_oRlRl and that of the round-leaf becomes O_nO_nrlrl. The F_1 generation from a cross of these two types is intermediate (O_oO_n *Rlrl*) and the F_2 generation falls into six phenotypic classes as shown in the following table:

Phenotypes	Theoretical Ratio and Genotypic Constitution	Observed Numbers*	Calculated Numbers
Okra	3 { 1 O_oO_oRlRl / 2 O_oO_oRlrl	21	14.82
Intermediate Okra	6 { 2 O_nO_oRlRl / 4 O_nO_oRlrl	30	29.64
Okra-round	1—1 O_oO_orlrl	5	4.94
Intermediate Okra-round	2—2 O_nO_orlrl	9	9.88
Broadlobed or Normal	3 { 1 O_nO_nRlRl / 2 O_nO_nRlrl	11	14.82
Round	1—1 O_nO_nrlrl	3	4.94
		$X^2 = 4.407$	
		$P = .30—.50$	

* Data from Brown and Cotton.

The numbers of plants in each class, taken from the data presented by Brown and Cotton, do not differ significantly from those which were expected from the proposed hypothesis.

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DIFFERENCES IN HETEROSIS OF WALNUT HYBRIDS

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It frequently has been reported that hybrids between Persian walnuts (*Juglans regia* L.) and various black walnut species which are known as Paradox hybrids, are uniformly very vigorous in growth. Below is presented a limited amount of data showing that this is not always true, but that seedling Paradox hybrids from a cross between certain parents are dwarfs in comparison with the progeny of another Persian walnut × black walnut cross.

In pollination work with the Franquette variety of Persian walnut, black walnut pollen from two sources was used in the season of 1931. One of the *J. nigra* parents was a grafted tree of the Stabler variety brought from Maryland. The other, known only as *Juglans nigra*, was an old black walnut tree growing in the Williamette valley of Oregon, whose only merit was lateness in shedding pollen.

A few nuts from these two crosses were saved and planted in order to grow seedlings with known parentage of the so-called Paradox hybrids. From harvest to planting time both lots of nuts were handled in the same manner and according to usual nursery practices. In the nursery, the nuts were planted in very rich river bottom soil where they

have remained. Other walnut nursery stock planted in the same location grows very vigorously.

Measurements of the total length of the shoots, taken at the end of each growing season after the leaves were shed, are given in the accompanying table for 1933, 1934, and 1935.

These data show that the Franquette × Stabler hybrids made good shoot growth and were vigorous, while the Franquette × *Juglans nigra* seedlings made poor growth and were low in vigor. This means that there is a genetic difference between the varieties of black walnut used as pollen parents. The Stabler pollen contributed to the hybrid factors for vigor and good shoot growth while the *J. nigra* pollen failed in this respect.

TABLE I. Total lineal shoot growth (mm.) of 2 Persian walnut × Black walnut hybrid progenies, showing difference in vigor.

Franquette × *Juglans nigra*				Franquette × Stabler			
Tree No.	1933	1934	1935	Tree No.	1933	1934	1935
1	19	92	406	1	156	225	432
2	1	78	320	2	95	317	1,684
3	31	72	361	3	138	345	2,200
4	17	130	470	4	157	352	1,316
5	66	100	983	5	158	500	1,430
6	21	135	1,118	6	182	540	1,537
7	34	232	899	7	60	212	856
8	92	282	1,384	8	33	220	1,186
9	44	57	328				
10	23	142	229				
11	72	175	559				
12	38	48	241				
13	49	122	493				
Average length	40	120	599		121	339	1,329

The Franquette × *Juglans nigra* hybrids averaged later in leafing out in the spring than did the others. In 1936 it was noted that when the Franquette × Stabler seedlings had new growth a foot or more in length, some of the Franquette × *J. nigra* hybrids had not even opened their buds. At the end of the growing seasons of 1933, 1934, and 1935 the Franquette × *J. nigra* trees shed the leaves much earlier. This may have been caused in part by a form of leaf scorch which was common to this group.

RECENT ADVANCES IN CYTOLOGY

THE second edition of *Recent Advances in Cytology* follows the same general outline as the first, with the exception of the last chapter. The book is devoted almost exclusively to the structure, morphology, and behavior of the chromosomes in relation to heredity. The first few chapters deal with mitosis, followed by descriptions and the interpretation of meiosis, structural hybrids, polyploidy, cytogenetics, and cell mechanics. The tabular summaries of various topics are useful, although a few are not very critical. In general, however, the presentation of material is an improvement on the first edition, and the interpretation of data appears to be more logical. The plates are much better than those of the first edition, owing to the excellent photographs provided by La Cour and Osterstock.

The last chapter of the original text has been omitted in the second edition. In the foreword of the first edition, Haldane wrote: ". . . I am convinced that the last chapter is a prolegomenon to every future theory of evolution." It is unfortunate that this masterpiece of mythogenesis is omitted from the new edition, but it has been replaced by another of even greater value, entitled, "Cell Mechanics."

Cell mechanics, including "internal mechanics," "external mechanics," and "ultra mechanics," deals with the forces responsible for the movement and relationships of the chromosomes during mitosis and meiosis. The analysis ap-

DARLINGTON, C. D., *Recent Advances in Cytology*. 671 pp. 2d Edition. Philadelphia: P. Blakiston's Son & Co. 1937.

pears to be logical only because all evidence contrary to the general theory is omitted. No attempt is made to explain chromosome behavior in the protozoa, the intranuclear spindles in the fungi, or meiosis in Sciara. After referring to Lillie's theory of electrical repulsions between chromosomes, and Kuwada's analysis of Meyer's floating magnet interpretation of chromosome orientation, Darlington concludes: "The spindle, as we saw, develops its molecular orientation under the influence of an electrical field and has the character of a liquid crystal." Some of the conclusions may be essentially correct, but they are not derived by the Newtonian principles advocated by the author. The integration of theories instead of the integration of data seems to be the keynote of this book.

Much of the material presented in the book is based upon the excellent cytological work of Darlington and his associates at the John Innes Horticultural Institution. Other work is included if it bears favorably on the theories being developed in the various chapters. In general, the author is discriminating in the selection of material, and irrelevant or erroneous contributions are omitted; but the same discrimination should have been applied to his own work. His 1929 paper on Tradescantia is frequently quoted, but with the exception of the chromosome counts, few of the numerous conclusions are correct.

The observations which conflict with Darlington's theories are lightly dismissed as optical illusions. But the author is impartial, for when his own observations do not agree with his theory, they also are ignored. Where no visible evidence is found to support a theory, the author is logically consistent and assumes that the structure must be present whether "visible or invisible" (p. 399).

The interpretation of other cytologists' observations is at times misleading. This applies especially to McClintock's ring chromosomes in Zea, Beadle's polymitotic Zea, and Nebel's work on the time of chromosome splitting in Tradescantia. Nebel was well acquainted with spiral chromonemata long before Darlington ever realized their significance. The discussion of the permanence of centromeres (p. 559) includes no reference to essentially the same conclusions reached by Morgan in 1926 and, in more detail, by Navashin in 1932.

The treatment of statistical data conforms to the usual standard of cytologists. One rather important conclusion is based on a total of 32 observations, covering 17 possible classes, and because the mode is slightly below the theoretical mode—a normal trend in segregates from triploids—the author assumes that there is a selective survival of segregates with 34 + 7 chromosomes in the progeny of triploid apples (p. 326). Although many of the conclusions are presumably based on quantitative data, there is not a single test of statistical significance in the entire book, and some conclusions appear to be reached without the aid of any data.

Belling, in reviewing the first edition of Darlington's book, suggested that a second edition would be improved by omitting nine-tenths of the numerous conjectures. The quota of conjectures does not appear to be diminished in the new edition. Those which have proved to be wrong have been replaced by new ones, apparently on the assumption that if enough are proposed, some may be correct. This is called the "deductive method" (p. VI).

The most important theory which has survived to be included in the second edition, is the confirmation of Janssen's hypothesis regarding the relation between chiasma formation and crossing over. Most of the erroneous conjectures of the first edition have been quietly withdrawn, including the *de novo* origin of the centromere. Of the conjectures in the second edition, some are obviously incorrect, and some can not be subjected to a critical test, but it is probable that more than 10 per cent may prove to be correct.

In a short appendix Darlington presents his views on the interpretation of chromosome structure and behavior. He quotes Bacon to the effect that science can progress only by the slow accumu-

lation of miscellaneous facts from which general laws may be derived in due time. Evidently the author overlooked a more appropriate quotation. Bacon also said there are two kinds of scientists, the ants and the spiders. The ants are the men of experiment who collect facts and use them. The spiders are the men of theory who spin cobwebs from their own minds. Many of Darlington's mental cobwebs are well constructed, and they serve a very useful purpose in outlining the design for further research problems in cytology.

Recent Advances in Cytology can not be recommended as a textbook for beginning students in cytology. The intellectual level is beyond the average undergraduate, who would have difficulty in distinguishing between theory and facts. For research workers and advanced students, the book is indispensable.

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THE INFINITELY SMALL

DURING 1936 the Rockefeller Foundation gave away over eleven million dollars "to promote the well-being of mankind throughout the world." The Foundation works on a very broad front, preferably through existing agencies. These comprised in 1936 41 local and national governments, 44 educational institutions, 20 research institutions, 2 libraries, 23 councils, associations, societies, and commissions, etc. Included in this budget of advances in human well-being are several research projects in genetics, through the Galton Laboratory of the University of London, and the Institute of Human Genetics of the University of Oslo. Research in mental hygiene, some of it bordering on human genetic studies, is also liberally supported. The natural sciences, which included the studies noted above, were appropriated $1,370,350 in 1936.

It is worthy of note and very encouraging that in this report the gene takes a much larger share of the total pages than gene-research takes of the year's total budget. Commenting on the infinitely small, Dr. Raymond B. Fosdick, president of the Foundation, has this to say about genes and other catalyzers of development and function:

> Some of the most dramatic developments which have come from experimental biology in the last quarter century are those tending to show the importance, in terms of human welfare and behavior, of substances so minute in quantity that only the most exact and delicate techniques of science are able to identify and measure them.
>
> Foremost in this catalogue of the infinitesimal stand the genes. So small are they that presumably no microscope can see them; they must be measured in terms of their effect. And yet the genes are the cargo ships of heredity, carrying compacted in their minuteness the whole load of inheritance from one generation to the next. Our bodily structure, the color of our eyes, our immunity to disease—all except that which we gain from environment—were potential or inherent in the genes of two parents. It has recently been estimated that if all the human sperm cells which are to be responsible for the two billion individuals who will constitute the next generation were gathered together they would occupy the space of half an aspirin tablet. If, of the corresponding egg cells, only the nuclei, which carry the stuff of life, be taken, they too would occupy the same space. It may be said, therefore, that the original and essential substance for the development of two billion individuals could be contained in a capsule no larger than an aspirin tablet.
>
> Equally small in quantity and extremely powerful in their influence are the hormones, chemical regulators secreted by the ductless glands. A third classification in this list of the infinitesimal would be the enzymes, which constitute another important group in the chemical machinery of the body. Scientists in many countries are now exploring the functions of hormones and enzymes; are learning to identify and isolate them; are beginning, in some cases, to produce them synthetically.
>
> In this whole area of the infinitesimal, experimental biology is faced with the necessity of doing quantitative analyses of an uncommon delicacy and accuracy. The microscope and ordinary chemical analyses are not enough. The exact techniques developed in studies of the inorganic are being adapted to the study of the organic.

Micromanipulators enable operations to be performed within a single cell. Protoplasm is being subjected to the terrific forces of the ultra-centrifuge in order that information may be obtained as to its molecular structure; the penetrating eye of the spectroscope is being turned from the stars to the blood stream; even the skill developed in radio engineering is employed when science needs to raise infinitely weak energies to the perceptible level.

The problems in this field are complex and difficult, but there is evidence of definite headway. Balanced against this are many failures and disappointments. In this, perhaps even more than in other scientific fields, there is no easy path, no promise of immediate and revolutionary results. Medicine is not yet ready to describe an emotion in terms of a chemical reaction or prescribe a hormone for a mental state. Press announcements, simplified for the lay public, sometimes encourage an optimism which is unjustified. The problems involved in this field require patience and a long look ahead. They constitute a relatively new and important factor in man's struggle to become a rational animal.

During 1936, the Foundation gave assistance in an amount approximating $900,000 to work in many aspects of the field of experimental biology of the type just outlined. In this brief report, it is possible to mention only one group of grants, those in support of research into the nature and function of the endocrine or ductless glands.

Studies of the Ductless Glands

"We are what we are, bodily, mentally, sexually, emotionally, facially, largely through the balance or imbalance of certain secretions discharged in minute quantities into the blood by the ductless glands." This generalization, by an eminent authority, outlines the field and something of the problem of endocrinology. Although certain phases of it were understood even by the early Romans, the modern study of the subject is about forty years old, and the greater part of the advance has been made in the last twenty years. The field, therefore, in comparison with other sciences relating to medicine, is relatively new and undeveloped.

All sorts of unanswered questions lie before the investigator. To what extent is old age determined by hormone factors? What could be done for hormone control to delay its approach or mitigate its effects? What effect does parathyroid deficiency have upon the amount and composition of the bile? Is our incapacity to cure childlessness in some cases due to an ignorance of endocrine factors? To what extent can the development of the brain be stimulated by hormone control? How far will we be able to modify character habits through the application of the techniques of this new science?

In a recent report by a special committee of the National Research Council under the chairmanship of Dr. Walter B. Cannon of Harvard (prepared for The John and Mary R. Markle Foundation), the following experiment is described, bearing on the last question in the foregoing paragraph. A rat, at first completely oblivious to offspring that may be offered to her for adoption will, after a few injections of prolactin (a pituitary hormone), eagerly adopt and mother as many as may be placed in the cage with her. Her maternal yearning seems to become universal. She will cherish not only infants of her own species but baby mice, baby rabbits, or, indeed, even baby squabs. "For a healthy adult rat," says the report, "to do other than make a prompt meal of the proffered squab betokens a fundamental change in her disposition. What is the explanation of the change? The reacting organs are the same; the energy of the system is the same; yet the reaction is strikingly different. We seem to be introduced here to what may well prove a far reaching biological principle that may be designated as 'chemical conditioning.'"

* * *

Renewing a contribution which had been given annually for a number of years the Foundation, in 1936, appropriated $75,000 to the National Research Council toward support of the work of its Committee for Research in Problems of Sex. These funds are, as in the past, allocated by the Committee to research projects which it will itself select. The Committee, made up of members of the faculties of Harvard, Rochester, Chicago, Johns Hopkins, and Yale Universities, represents varied approaches to the problem. Beginning work in 1920, it has demonstrated what careful planning can accomplish in a formerly undeveloped field of research.

In all this work, science is standing on the shore of a new continent. By and large, our knowledge of the control of hormone secretions is in a fragmentary state. What lies ahead, what secrets of human well-being may be discovered, what techniques for the shaping of human personality may be developed only time and long patience can tell.

As the promotion of the well-being of mankind becomes more science and less hopeful guess, intensive consideration will have to be given to Mr. Fosdick's "aspirin tablet" of gigantic portent, which encompasses so neatly the chromosomal heritage of the entire human race. Insignificantly small, it is nevertheless a rock on which poorly conceived plans "for the promotion of well being" will inevitably be shattered. At the same time it is potential-

ly capable of serving as an adequate foundation on which a comprehensive, sound, and basically humane structure of human betterment may be reared. Aspirin tablets should dissolve quickly, the radio announcers tell us, so that they can the sooner go about preventing and curing headaches. Here is a tablet whose solution, could it be accomplished, might go far toward curing the headaches of philanthropists, statesmen, and of men of good will generally, for all time.

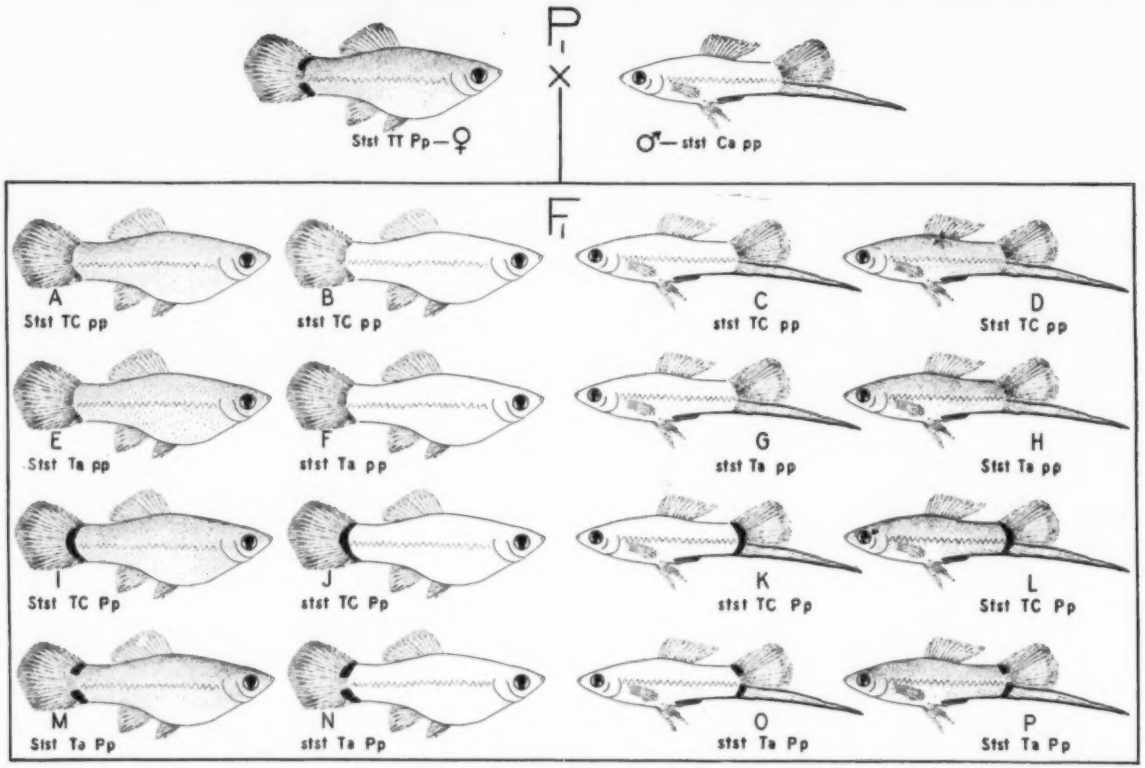
But alas, this tablet is disconcertingly hard to dissolve. Its properties are so vast and complicated that they escape us. Since the analysis still defies our ingenuity, we are inclined to forget the aspirin tablet altogether, and to assume that it has chameleon-like properties—capable of being anything to all people. Unfortunately there is no justification for such a hope, and before we can take many constructive steps to advance general welfare we must know just how our aspirin tablet is put together, and just what we can do to arrange human society to fit our tablet, and what can be done to make the two billion distinct ingredients in our tablet fit into the human society of our dreams. How does the human aspirin tablet react to mass education? To mass suggestion? To what extent do all human aspirin molecules react the same and how do they react differently? The final answer to these questions and a multitude of others lie partly in this field of the infinitely small, an unknown land which challenges the explorers of the future.

"THE BLOOD GROUP OF DIPLOMACY"

AN Associated Press report, from Tokyo on June 24, states that Dr. Isunemasa Niigaki, Medical Advisor to the Foreign Office, suggests that diplomats be picked by blood groups types,—the "Q" group of the international nomenclature showing outstanding diplomatic properties according to his report. Only "superior men," like Premier Konoya, who fortunately is of this blood group, are held to be suitable to represent Japan among the nations.

If Dr. Niigaki has valid evidence to prove the existence of the "blood group of diplomacy," this will represent the first case in which linkage between one of the blood groups and any morphological, physiological, or psychological character has been discovered. The failure to find such linkages is not due to lack of effort, for the literature of the subject abounds with observations and statistical studies directed toward this end. Many alleged correlations have been suggested, but detailed analysis has shown all of them to be without foundation. This is all the more remarkable because a difference in blood reaction of such magnitude might well be expected to be correlated with resistance to some of the host of diseases which plague mankind. No significant relation of such a character has been accepted by competent blood group researchers.

Dr. L. W. Parr, of the Medical School of George Washington University, an authority on blood groups, has kindly made available to the writer a little data on this point. Dr. Parr has compiled records of the blood groups of all medical students at George Washington University in recent years. Thirty students, with an average grade of "A" in his classes, are distributed as follows: 12 *O*, 12 *A*, 5 *B*, and 1 *AB*. The distribution among 426 medical students is: 45 per cent *O*, 38.5 per cent *A*, 12.1 per cent *B*, and 4.2 per cent *AB*. This is approximately the distribution in the general population of the United States. Thus this small sample, as far as it goes, does not confirm Dr. Niigaki's announcement that a particular blood group is associated with mental ability. It might be argued that outstanding attainments in diplomacy involve a special ability different from the intelligence which produces "A" medical grades. This might be associated with the *O* blood group as alleged, but no confirmatory data on this point seem to be available.—R. C. C.



THE MATING OF A COMMON, TWIN-SPOT, SWORDTAIL WITH THE GOLD

Figure 13

This diagram illustrates one of several results obtained when a twin-spot swordtail is mated with one without a pattern near the tail region. It also illustrates the independent hereditary behavior of the character "stipples", the presence of which characterizes the common, green or wild type swordtail and the absence of which characterizes the golden swordtail. In this instance the female swordtail is stippled, *St* and twin-spotted *T P* while the golden swordtail male is recessive, non-stippled, *st*. This golden male is dominant for the factor *C* for crescent but it does not show the crescent pattern because it is recessive for a pattern forming accessory factor *p*. From the breeding results the genotype of the male is *stst Ca pp;* the female is *Stst TT Pp*.

HERITABLE COLOR VARIATIONS IN THE MEXICAN SWORDTAIL-FISH

Aquarium Species as the Drosophila of Fish Genetics

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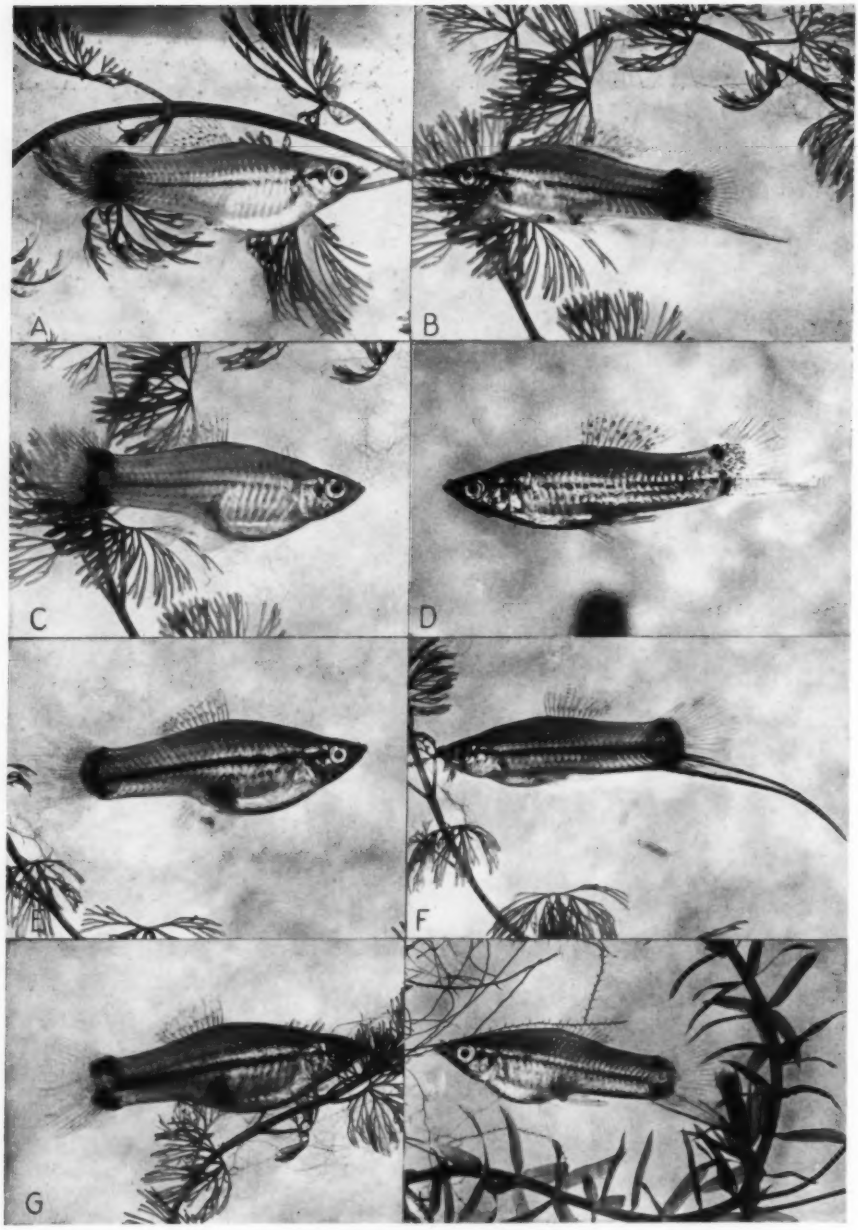
THE study of the genetics of fishes has progressed steadily since Goodrich[9] reviewed Mendelian inheritance in fishes in 1929. Unfortunately only a few workers, less than half a dozen, are engaged in these studies in America. Practically nothing is known in a modern genetic sense of the inheritance of the most obvious variations among our commercial and game fishes. This is regrettable, since genetic studies with aquarium fishes indicate that these forms are subject to the same fundamental hereditary principles that apply to other animals and plants. The culture of game fishes is a group industry, but as a group those engaged in this work are far behind —indeed it may be said truthfully that they have hardly started—in the profitable utilization and application of genetic principles for the improvement of breeding stock. For the time being, examples of heredity in fishes must be obtained from the small aquarium species. These may in time serve the game fish culturist in the same useful manner that the ubiquitous fruit flies, the despised rats and mice, and the patient guinea pigs, have served mankind in general and the animal husbandryman in particular—when these small and conveniently cultured animals are confined and studied in the genetic laboratory. The author[11] has already sketched the usefulness of some of the small fishes in the world of science.

The Mexican swordtail is a common tropical aquarium fish. It has held its popularity among fish fanciers and amateur aquarists ever since its introduction into Germany in 1909. The first living specimen, a female, was taken to Germany from Puerto Barrios, probably from one of the small tributaries of the Rio Coatxacoalcos in the extreme southern part of the State of Vera Cruz. Very soon after the first female specimen was received in Germany several more fish were shipped. In this second lot a male was included, but the locality from which these fish were taken is not stated by Arnold,[1] the first writer to chronicle the arrival of the swordtail in Germany.

The first living swordtails to enter the United States came from Germany, about 1911, and were probably the offspring of those early Mexican fish immigrants to Germany. The swordtails followed their close relatives, the Mexican platyfish to the United States.[13]

In 1926 the aquarium bred swordtail had nine color varieties.[30] There are many more today.[24] How many of these have representatives in the feral state in Mexico is not known, but it is fairly certain that not many varieties exist there. Not one of the brightly colored strains in the class of red, orange, yellow or whitish green has been mentioned by collectors. If these represent wild type forms they have not yet been seen. In three recent trips to Mexico and Guatamala by Dr. C. L. Turner, Dr. C. L. Hubbs and the author, no red, yellow or albinistic forms were seen.[18a]

A detailed study of the history and origin of all the varieties of swordtails is now being undertaken. The present paper will outline the mode of inheritance of a number of well established patterns without regard to their origin. Many strains of aquarium swordtails have had their origin by hybridization with the platyfish, *Platypoecilus maculatus*. Some may have originated by hybridization with its co-species, *Xiphophorus montezumae* of the Rio Panuco System.



SWORDTAIL PATTERNS

Figure 16

Females on left and males on right, showing four pigment patterns. *A, B,* show the gold crescent pattern (*st CP*). *C-D* shows the gold twin-spot pattern (*st TP*). *E-F* shows the stippled crescent pattern (*ST CP*). *G-H* shows the stippled twin-spot pattern (*St TP*). The common green wild type swordtail and the forms showing micromelanospores (small black spots) over the body carry the *Sp* factor. The gold form has no black pigment spots on the body; it carries the recessive allelomorph, *st*. For explanation of relation of genes *C P, TP, St* and *st, CP* and *Cp* see page 229 and Table III.

Types of Swordtails

a. The Common, Green or Wild Type Swordtail

The so-called green swordtail is regarded as the wild type. It is characterized by an olive green color with one prominent red zigzag lateral stripe. The olive green appearance is due to the presence in the skin of the swordtail of many small black pigmented cells or micromelanophores (Gordon[6]). The absence of these micromelanophores makes the swordtail appear golden in color. A parallel condition exists between the olive gray-green (stippled) platyfish and the gold platy (Gordon[15]).

Two substrains of the green swordtail are recognized by fanciers. They exist in the wild state in Mexico. One strain has a green "sword" while the other has a golden "sword." Those strains which have been a long time under domestication usually have a green sword. At Jalapa in Vera Cruz, in a pond outside the city limits, the swordtails have golden swords preponderently.[15] The genetics of sword coloring has not been studied critically.

b. The Golden Swordtail

The golden swordtail is characterized by a yellowish orange body with a red zigzag lateral stripe. The golden swordtail is golden because it has lost the innumerable small black pigmented cells in its skin covering. These black cells are present in full complement in the green swordtail. Their presence in the green swordtail depends upon the action of an autosomal gene *St*, and their absence in the golden swordtail (rarely are the black cells entirely absent) depends upon a recessive allelomorph *st*. Kosswig has shown that in the swordtail, the micromelanophore factor in crosses between green and albino (described below) follows the behavior of the micromelanophore factor in crosses between the stippled and gold platyfish. This paper will show that the micromelanophore factor behaves in the same manner in crossing green and golden swordtails.

The first definite mention of the gold swordtail in the literature of the aquarium was made in 1921 by Krasper.[21] Its origin is attributed to a sport: a xanthic form appeared from stocks of green swordtails. Fritz Hildebrand, observing the life in his community aquarium, noticed that "The stately Xiphophorus male (green) mated with a golden female of the same species and from this alliance a number of young were produced which later resembled the green father." [17] This observation is the first concerning the dominance of green over gold. A preliminary statement of crosses between green and gold was given by Gordon[15] in 1934.

c. The Albino Swordtail

The written records show that the albino swordtail appeared both in the United States and in Germany in 1934. In America the first albino swordtails were developed by the late Dr. August Traeger of Brooklyn, N. Y. Mystery surrounds the history of the albino's discovery as it does all new varieties. Peter's unconvincing story[23] of Dr. Traeger's "method" of developing the albino strain is inadequate. Mrs. Traeger recalls only that the first albinos appeared in a group of four in a brood of green swordtails, from specially selected green swordtail parents. The characters selected were not divulged.

Curiously enough in Germany about the same time, April 17, 1934, the veteran aquarium reporter, Wilhelm Schreitmüller, announced the appearance of a *"Totalalbino von Xiphophorus helleri."* It turns out that a member of the *Franzensbader Vereins* in breeding a normal green swordtail obtained 150 young, four of which, two pairs, were albinos. Schreitmüller is told that these albino females produced only normally colored offspring. It is on this basis that Bailey[2] claims that these German albinos swordtails were not genuine. But it is entirely possible, indeed probable, that these albinos did not breed true because they were not segregated and were undoubtedly fertilized with sperms coming from green colored swordtails of the same brood. This record of albinos in Germany may be accepted as valid.

Apparently the albino swordtail in Germany was developed much earlier than the date, April, 1934, because Schreitmüller quotes from a letter re-

ceived by Prof. Dr. C. Kosswig of the Natural History Museum of Brawnschweig, under the date of Nov. 27, 1933, in which Kosswig thanks Schreitmüller for his kindness in presenting him with an albino and says that it will be an extremely useful type for his study of inheritance. It was from this original stock that Kosswig produced the many fishes he writes about in his 1935 paper. The story was reviewed by Breider[3] in 1935.

Mr. C. W. Coates of the New York Aquarium, writing July 7, 1934, in the New York *Sun*, said that Mr. Traeger had a fairly large stock at the time of his interview. Mr. Coates estimates that there were not less than 2,000 albino specimens, indicating that Traeger's fish were developed over a number of years, perhaps as many as 15, as Traeger claimed.

d. The Twin-spot and the Crescent

Two years after the first swordtail was brought to Germany a new variety of the swordtail arrived from Guatemala. The arrival of the twin-spot swordtail in 1910-1911 was destined to arouse a controversy which in the minds of many aquarists is not yet settled. In 1911 Regan of the British Museum wrote under the new species of swordtail he had just named, *Xiphophorus rachovii:* "Six examples of this pretty little aquarium fish have been sent to me by Herr A. Rachow of Hamburg (Germany). They come from Porto Barrios on the Atlantic coast of Guatamala between Lake Yzabal and Rio Montagua." These fish had a pair of conspicuous spots, one above and one below, at the base of the caudal fin. Two years later, in 1913, Regan revised the taxonomic relationships of these fishes. He found that other specimens from Puerto Barrios, received later, lacked the spots of the original forms. He decided that *X. rachovii* was not a new species. He ended his statement with these words, "Whether this (twin-spot type) is another variety of *X. helleri* or a hybrid with *Platypoecilus maculatus* as some aquarium writers in Germany believe, still remains to be settled." The first account of *X. rachovii* as an aquarium fish was written by Rachow[25] in 1911.

Since Regan's statements cast doubt upon the validity of the twin-spot pattern as a pure swordtail character two schools of thought developed in the ranks of German aquarists. Those who believed that the twin-spot pattern was entirely a Xiphophorus character and that the imported forms had this character upon arrival wrote their stories in the *Blätter fur Aquarien und Terrarienkunde.* Those who believed that the "rachovii" pattern was a synthetic pattern derived from hybridization of a swordtail with a platyfish that had this pattern, wrote their stories in the *Wöchenschrift f. A. u. T.* Many elaborate experiments were cited to show how a character from one species may be transferred to another.[22] The experiments of Gerschler[4] are outstanding but he used the *"halbmond"* platyfish pattern—a filled crescent pattern as illustrated recently by Gordon.[8, 15] This is different from the twin-spot. Gerschler produced hybrids with the *"halbmond"* but no one ever described the mechanics of the transfer of the twin-spot of the platy to the swordtail. Some have suggested that Xiphophorus and Platypoecilus with a twin-spot may have crossed in Guatamala, but the only collections ever made of the platyfish were made far in the interior—not at Puerto Barrios.

The first collection was made in Mexico, near Uaxactum, by Van Tyne.[18] Professor C. L. Hubbs collected the second group of 633 platyfish in Mexico from Lake Peten in central Guatamala but none from Lake Yzabal near the coast. Furthermore, of the 636 platyfish from Guatamala, only nine have the twin-spot or the "rachovii" patterns, showing that this pattern is rare.

In the light of the fact that *Platypoecilus variatus* and *P. xiphidium* have twin-spot patterns, too, it is likely that the twin-spot pattern of Xiphophorus has been attained by the swordtails by natural processes of evolution.

The Genetic Behavior of Stipples (Micromelanophores) as Shown by "Green" × "Golden" Crosses

The first cross in Table I shows that the strain of golden swordtails was true

breeding. In crosses, 2, 3 and 4 when green and golden swordtails were mated, the results show the dominance of stipples over gold or the dominance of micromelanophores over their absence; all the 232 individuals were *St;* none was *st* (gold).

In a series of back-crosses of the heterozygous stippled-gold (*St st*) swordtails to the recessive *st st* golden swordtails, the parental types were recovered. The expected ratio of one stippled to one gold swordtail did not appear. The ratio appeared to be more of a 5:3 instead of the 1:1 ratio expected. The total number of offspring of crosses 5, 6, 7, 8 and 9 was 396, of which 249 were stippled and only 147 were golden. On the basis of the expected 1:1 ratio 198 individuals should have appeared in each of the two groups. These aberrant results are attributed to the well known semi-lethal nature of the recessive factor *st* for gold. (It may turn out that the gold factor itself is not a semi-lethal but that semi-lethal factors may be closely linked to the gold factor.) Kosswig has found the albino swordtails weaker at the early stages of development than the normal stippled type. His ratio in back-cross experiments is 77 stippled to 66 non-stippled. Yet it is held among fish fanciers that certain strains of gold swordtails are more vigorous than others. Perhaps these vigorous strains lack the actual semi-lethal genetic factors.

In two matings, crosses 10 and 11, when heterozygous stippled (*St st*) swordtails were crossed, a perfect 3:1 ratio was obtained indicating further that a single factor only is involved. In Kosswig's crosses involving eleven matings of heterozygous stippled-albino hybrids he obtained 446 stippled to 108 non-stippled swordtails. His interpretation is that there is one factor involved. This factor he designates as *P* and *p*.

The crosses between the golden and albino swordtails for the purpose of studying the genetic relationship of these two color phases have not yet been worked out—but the work is now in progress. Kosswig[20] and Breider[3] have indicated that the red and stippled swordtail when crossed to an albino produces red and stippled hybrids which when backcrossed to albinos produce red and stippled, stippled, red-albinos and pure white albinos.

The Inheritance of the Twin-spot and Crescent

Miss Kerrigan[19] was the first to show by controlled genetical methods that the twin-spot pattern in the swordtail is inherited autosomally. The twin-spot pattern is associated in heredity with the crescent pattern. The origin of the crescent pattern is obscure. It apparently developed from the twin-spot by a method of extension of the pigmented areas of the twin-spot so that the two areas of pigmentation were joined. Miss Kerrigan has shown that the twin-spot pattern may be referred to a dominant gene *C* (part crescent). A non-twin-spot phenotypically is *c*. The crescent pattern in the swordtail depends upon the presence of the twin-spot factor *C* and another dominant factor *P*, a factor that may act as an extensor of *C*. The *C P* combination produces the crescent. In her experiments *P* and *C* behave genetically as independent autosomal dominant factors as no linkage is shown. The possible combinations are *C P*, which is the crescent, and *C p*, which is the twin-spot; *c P* and *c p* produce no pattern.

From the point of view of comparative genetics it is important to bring out the fact that the platyfish has a twin-spot and a crescent pattern. While the twin-spot pattern appears to be homologous structurally with the twin-spot of the swordtail, the crescent of the platy, as indicated by many authors and described by Gordon,[8] 1931, is not entirely homologous with the crescent of the swordtail. The crescent pattern of the platyfish has a part which is not present in the swordtail—a part which Gordon[8] has described as a mark shaped like the head of an ancient battle-ax. Gordon described the inheritance of this crescent, *C*, and the twin-spot, *T*, in the platyfish, and concluded that they belong to a dominant autosomal allelomorphic series within which series also belong the factor *M* for moon pattern and the factor *O* for one-spot.

TABLE I. Crosses of Gold and Stippled Swordtails.

Number of Cross	Females			Males			Progeny			
	Color	Genetic Formula	Pedigree Number	Color	Genetic Formula	Pedigree Number	Culture Number	Stipple	Gold	Totals
1	Gold	stst	180-1	Gold	stst	180-11	180	-	86	86
2	Gold	stst	180	Stippled	StSt	109	181	108	-	108
3	Gold	stst	186-1	Stippled	StSt	119-12	186	13	-	13
4	Gold	stst	190	Stippled	StSt	109	190	111	-	111
							Total ♂	232	-	232
5	Stippled	Stst	188	Gold	stst	180	184	37	36	73
6	Gold	stst	180-2	Stippled	Stst	188-11	188	49	26	75
7	Gold	stst	180-4	Stippled	Stst	190-14	227	9	8	17
8	Gold	stst	180-5	Stippled	Stst	190-15	228	30	9	39
9	Stipple	Stst	190-3	Gold	stst	180-16	229	124	68	198
							Total ♀	249	147	396
10	Stipple	Stst	229-5	Stipple	Stst	229-13	235	68	22	90
11	Stipple	Stst	185	Stipple	Stst	229-14	249	8	3	11
							Total ♀	76	25	101

TABLE II. Crosses of Twin-spot and Crescent Swordtails.

Number of Cross	Females			Males			Progeny						
	Color Pattern	Genetic Formula	Pedigree Number	Color Pattern	Genetic Formula	Pedigree Number	Culture	Crescent Stipple	Crescent Gold	Twin-spot Stipple	Twin-spot Gold	No pattern Stipple	No pattern Gold	Totals
1	- Gold	cc Pp stst	180	Crescent Stipple	Cc PP StSt	109-a	181	52	-	-	-	56	-	108
2	- Gold	cc pp stst	190-c	Crescent Stipple	Cc Pp StSt	109-b	190	25	-	20	-	66	-	111
3	- Gold	cc PP stst	180-4	Crescent Stipple	Cc Pp Stst	190-14	227	4	6	-	-	5	2	17
4	- Gold	cc Pp stst	180-5	Twin-spot Stipple	Cc pp Stst	190-15	228	11	1	8	1	11	7	39
5	Twin-spot Stipple	Cc pp Stst	190-3	- Gold	cc Pp stst	180-16	229	26	14	34	16	64	38	192
6	Twin-spot Gold	Cc pp stst	229-1	- Gold	cc Pp stst	180-15	231	-	2	-	6	-	8	16
7	Twin-spot Gold	Cc pp stst	229-2	- Gold	cc Pp stst	180-15	232	-	15	-	6	-	15	36
8	- Stippled	cc PP Stst	185-a	Crescent Stippled	Cc Pp Stst	229-3	249	3	2	-	-	3	1	11
9	Crescent Gold	Cc Pp stst	249-1	- Stippled	cc Pp Stst	279-11	279	17	17	3	1	18	20	76

TABLE III. Genotypes of Crosses shown in Table II, according to alternative hypothesis.

No.	Females			Males		
1	Gold	CC pp	180	Crescent	CC Pp	109a
2	Gold	Tc pp	190c	Crescent	CT Pp	109b
3	Gold	CC pp	180-4	Crescent	Cc Pp	190-14
4	Gold	Cc pp	180-5	Twin	TT Pp	190-15
5	Twin	TT Pp	190-3	Gold	Cc pp	180-16
6	Twin	Tc Pp	229-1	Gold	CT pp	180-15
7	Twin	Tc Pp	229-2	Gold	CT pp	180-15
8	Stippled	CC pp	185a	Crescent	CT Pp	229-3
9	Crescent	CT Pp	249-1	Stippled	Cc pp	279-J

Note: fish listed as Gold or Stippled have no pattern like crescent or twin

But there is in the platyfish a fifth pattern, which is structurally homologous to the crescent of the swordtail. It was listed as having occurred among the specimens that Meek collected in 1904. It is listed as number *G* in Figure 2 by Gordon and Fraser, 1931.[16] No living fish carrying the *single crescent, C c*, were available until 1932 when Gordon collected a number of them in Mexico.[13] The genetics of this form has not been worked out.

The results listed in Table II were not obtained from systematic procedure or planned series of experiments. The swordtails listed in Table II represent various stocks that were used for the hybridization experiments. These results were obtained as a by-product from work on hybridization and its effect upon the state of melanosis in hybrids between the swordtails and platyfish.[9] Consequently there are many places where further work should have been done to clarify certain situations.

Since further work in this laboratory is not likely to be possible, these data are presented at this time. The nine crosses and their results listed in Table II may readily be explained upon the hypothesis which Miss Kerrigan proposed in 1934,[19] namely, by referring the factors *C p* to the twin-spot pattern, the factors *C P* to the crescent, and *c P* and *c p* to no factor for either crescent or twin-spot. This idea is carried out by designating the probable genetic constitution of the swordtails with the symbols proposed by Miss Kerrigan (see Table II).

If, however, we wish to assume that the twin-spot factor is allelomorphic to the crescent factor, an alternative hypothesis may be suggested which will fit all the data presented in Table II. This alternative hypothesis utilized in Table III is also effective in explaining all of Miss Kerrigan's 1933 data. Which hypothesis is correct the future must decide.

The alternative hypothesis states that the twin-spot may be referred to the factor *T* and the crescent may be referred to the factor *C*. These are allelomorphic. Neither *T* nor *C* is effective in producing a pattern without the presence of the factor *P*, for pattern formation. In this hypothesis the crescent pattern may be written as *CC PP, Ca PP, CC Pp* where *a* represents the recessive of the pattern or the absence of the dominant color factor. The twin-spot pattern may be written as *TT PP, Ta PP, TT Pp*. The genotypes *CT PP* and *CT Pp* will be crescent. A comparison of the two systems may be made by consulting Table III for the data listed in Table II.

Discussion and Conclusions

From the data listed in Table II it may readily be seen that there is no apparent linkage of the factors *C* or *T* with *St* or with *P* in the swordtail. Neither is there any indication of linkage of *C*, *T*, *O* or *M* in the platyfish with the *St* of the platyfish.[16]

In crosses between the spotted platyfish *Sp* and the unspotted swordtail *sp*, the hybrids come down with a neoplastic disease, melanosis. This abnormality may be stated to arise from the unrestrained growth of macromelanophores—the cells which the factor *Sp* controls. But the swordtail contributes the excitation factor which induces macromelanophores to unrestrained growth in the hybrids.[9, 10] Whether this excitation factor is the *P* of the swordtail has not yet been shown. It has definitely been shown that while the factor *St* of the platyfish increases the growth of macromelanophores (*Sp*) in the platyfish and this is also characteristic of the *St* of the swordtail—it is neither the *St* of the platyfish nor *St* of the swordtail that brings about melanosis in the hybrids between the platyfish and the swordtail.

The interpretation that *C* and *T* are allelomorphic in the swordtail is useful but not essential to show the intimate relationship of this species with the platyfish where many caudal peduncular markings may be referred to factors that behave as dominant allelomorphs of each other. It would be valuable to know if the twin-spot and the crescent of *Platypoecilus variatus* and *Platypoecilus xiphidium* belong to an allelomorphic series. These two species show parallelism in these patterns.

Summary

1. The stipple effect in the swordtail seen in the common, green or wild type is due to the presence in the integumentary tissues of many small pigment cells or micromelanophores.
2. The stipple effect is dominant over non-stipple—the non-stippled fish is gold in color. The factor is *St* and *st*.
3. The twin-spot and crescent patterns in the swordtail may be referred to the factors *C* and *P* where *C P* is crescent, *C p* is twin-spot, *c P* and *c p* are neither crescent nor twin-spot. This is Miss Kerrigan's interpretation and is suitable to explain the data presented in this paper.
4. The twin-spot pattern may be referred to the factors *T P* and the crescent to factors *C P*. The factors *T* and *C* are allelomorphic to each other and to the recessive indicated by *a*. An *a P* or *a p* would indicate no crescent and no twin-spot. *CT P* is crescent. Item number 4 is the alternative hypothesis proposed in the paper.

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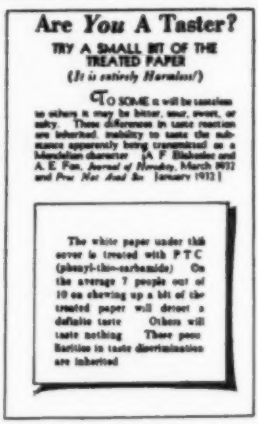
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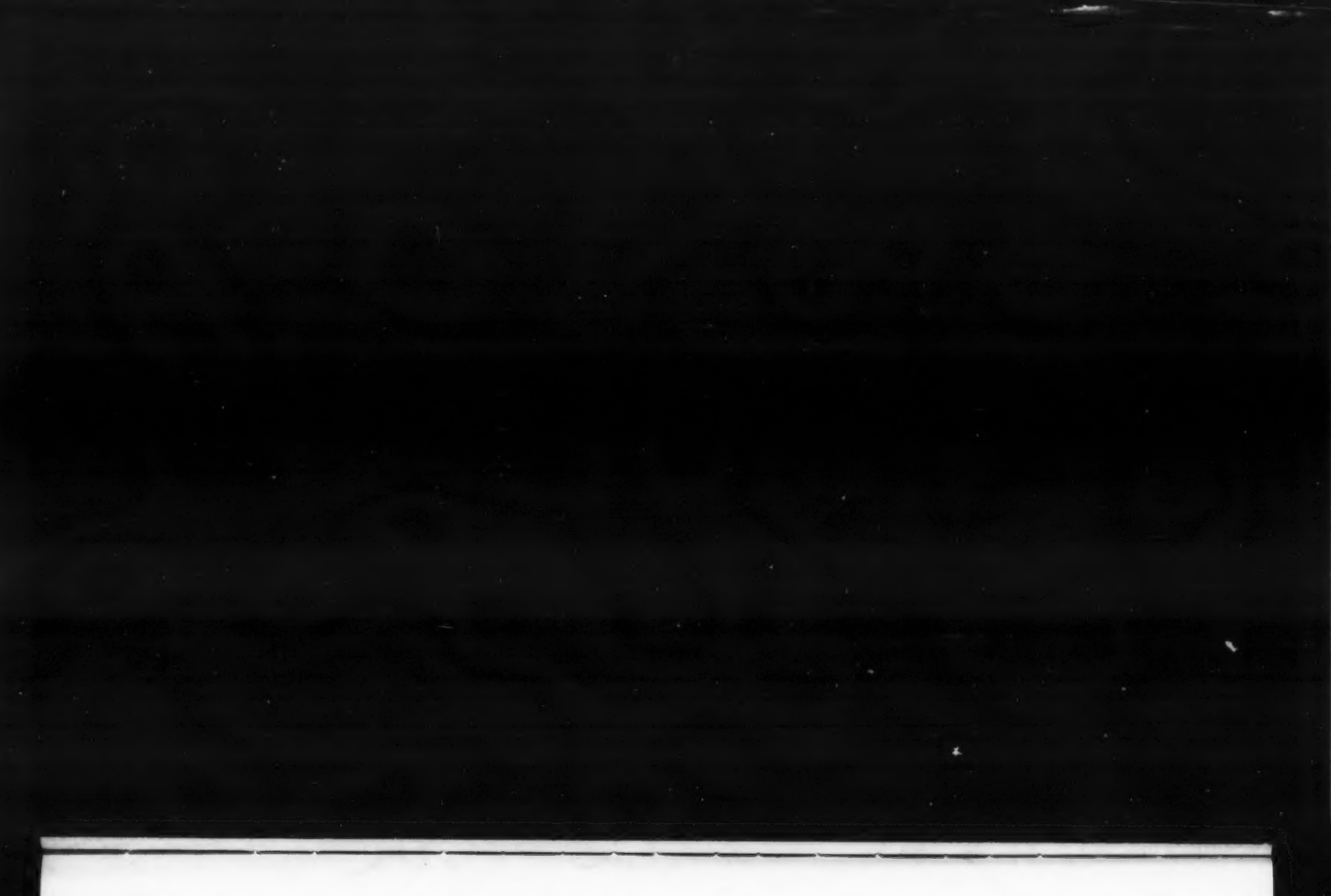
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